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Stk. ed. 1912.
Kl. 2.

Oslo. Stk. ed. 1912
2nd part

Text-Book.

~~In all the infections~~

In many of the infectious diseases there are figures of no. of cases + deaths. Should we change these to the latest yrs published?

Typhoid Fever. Should we try to add something additional about Paratyphoid.

p. 43. The section (c) on Serum Therapy should be changed.

Septicaemia. should we try to add a note as to focal infections, especially of the teeth?

Diphtheria. a note regarding the Schick reaction?

The local treatment (p. 71) might be condensed?

Pneumonia. a note as to the types? and the serum treatment (p. 79, 100)?

The chart on p. 82 seems of doubtful value.

Broncho-pneumonia. The primary form is not uncommon in adults. This should be mentioned as the text suggests that it occurs only in children. Please note this for the revision.

Whooping cough. organisms in diagnosis? vaccine + serum in treatment?

Tetanus. under treatment combination of intraspinal + subcutaneous intravenous injections?

(The results in N.Y. are striking. They give

3000 - 5000 units intraspinally + repeat in 24 hours

1000 units intravenously

10000 " subcutaneously in 3 or 4 days)

Tuberculosis. Should there be a note on the "acute tuberculous septicaemia" as described by Landouzy?

Should there be a note as to the morbid anatomy of the general + pulmonary forms of miliary tubercle? This done for the meningeal form.

Under the diagnosis of pulmonary tuberculosis should there not be a note about the recognition of cachexia + other conditions where the signs are marked (such as the mycoses, Syphilis etc)?

Yes

Syphilis – we should add the adenitis under secondary stage.
of nervous system – are there any microscopic changes?

~~Anemia~~ Should there be some mention of pain?

Typhus fever – the question of abscesses?

Polio-myelitis – I suppose this should be shifted to diseases of known etiology.
It may not be possible to do this now.

Pellagra – I suppose there should be added the diet (protein) view of
the U.S. Public Health service.

Diabetes mellitus – The treatment should be revised. You mention, there is a
letter to Dubelles you might have in mind.

Stomach – I suppose this whole section should be recast. This can only be
considered for a full revision.

Constipation (p. 546). Do you wish to make any changes in the statistical
regarding x-ray studies.

~~p 644~~ ~~Is pneumonokoniosis or pneumokoniosis the better form?~~

p. 698. A student sent in the query if "anaemia" was correct as the
first word of paragraph 3 under Symptoms. He asked if it should not
be oedema – see the remainder of the paragraph. In its present form
it has survived from the first edition.

Acute nephritis. The acute exacerbations in chronic nephritis should be
mentioned under Diagnosis.

Chronic nephritis. no change at present I presume – but what can we do to do in
the revision?

p. 718. The statement also constituents of calculi should be altered. Is it
not doubtful if uric acid are the most important?

Pernicious anaemia. under treatment - some mention of splenectomy.

p. 745. The no. of leucocytes should be mentioned under lymphoid leukaemia

Hodgkin's disease. what about the organisms question?

Purpura. what additions at present?

Arrhythmia. p. 771 et seq. Should there be any change here?

p. 845. Crisp's figures. Some one told me that there was a marked error in these figures but could not remember in what particular. You quote here a series of 501 cases; in the System, second ed. vol iv. p. 473, you quote 555 cases.

p. 865. under Thymus we should note the fact in the adult of persistent thymus as described by H. Simmons. (Arch. Int. Med. 1914, xiii, p. 169

p. 877. There is much Mayo clinic work on the gland in Graves's disease.

p. 885. Do you regard splenic anaemia and Banti's disease as synonymous terms? It is not very clear.

Newer Systems. I leave this to you to suggest changes. It is a problem. The newer things regarding syphilis should be mentioned. see also Renaturing atoxyl. p. 268.

Vagotony. etc.
Phlebectasia and arterial glyc. - Thromboangeitis etc.
Rat Bite Fever - change position
Poliomyelitis -
Ayaus -

tion is present, but almost all signs of exudation are wanting. Close inspection may be necessary to detect a slight dulling of the serous surfaces.

The amount of the effusion varies from half a litre to 20 or 30 litres. There are probably essential differences between the various kinds of peritonitis.

Bacteriology.—A large number of organisms have been found in connection with the disease. In Flexner's series, in 12 primary cases the streptococcus was the prevailing organism. In the cases following operation the staphylococcus was present alone in 12 out of 33, the streptococcus in 5, and the colon bacillus in 5. Other organisms were the pneumococcus, bacillus pyocyaneus, and bacillus aerogenes. Of 56 cases of peritonitis following intestinal infections, the colon bacillus occurred in 43, usually in connection with streptococci. The bacillus lactis aerogenes has also been found as the sole organism. The gonococcus is present in the form which arises from salpingitis and may occur in the gonorrhœal infections of children.

Much attention has been paid ~~of late~~ to the pneumococcus as an agent in the causation of peritonitis, and many cases are of the primary form without recognizable portal of entry; but ~~it is to be remembered that~~ there are many latent pneumococcic lesions, particularly those of the middle ear, and of the accessory sinuses of the nose. Cameron, ~~in a recent study,~~ makes two groups of cases; a diffuse form setting in with abdominal pain, high fever, vomiting, and diarrhœa, in which death may occur within 36 hours. In the other group the peritonitis is local, and the symptoms may suggest appendicitis. Gradually a localized abscess develops, which may rupture internally. The creamy greenish yellow odorless pus is very characteristic.

Symptoms.—In the perforative and septic cases the onset is marked by chilly feelings or an actual rigor with intense pain in the abdomen. In typhoid fever, when the sensorium is benumbed, the onset may not be noticed. The pain is general, and is usually intense and aggravated by movements and pressure. A position is taken which relieves the tension of the abdominal muscles, so that the patient lies on the back with the thighs drawn up and the shoulders elevated. The greatest pain is usually below the umbilicus, but in peritonitis from perforation of the stomach pain may be referred to the back, the chest, or the shoulder. The respiration is superficial—costal in type—as it is painful to use the diaphragm. For the same reason the action of coughing is restrained, and even the movements necessary for talking are limited. In this early stage the sensitiveness may be great and the abdominal muscles are often rigidly contracted. If the patient is at perfect rest the pain may be very slight, and there are instances in which it is not at all marked, and may, indeed, be absent.

The abdomen gradually becomes distended and tense and is tympanitic on percussion. The pulse is rapid, small, and hard, and often has a peculiar wiry quality. It ranges from 110 to 150. The temperature may rise rapidly after the chill and reach 104° or 105° F., but the subsequent elevation is moderate. In some very severe cases there may be no fever throughout. The tongue at first is white and moist, but subsequently becomes dry and often red and fissured. Vomiting is an early and prominent feature and causes great pain. The contents of the stomach are first ejected, then a yellowish and bile stained fluid, and finally a greenish and, in rare instances, a brownish

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black liquid with slight fæcal odor. The bowels may be loose at the onset and then constipation may follow. Frequent micturition may be present, less often retention. The urine is usually scanty and high-colored, and contains a large quantity of indican.

The appearance of the patient when these symptoms have fully developed is very characteristic. The face is pinched, the eyes are sunken, and the expression is very anxious. The constant vomiting of fluids causes a wasted appearance, and the hands sometimes present the washer-woman's skin. Except in cholera, we see the Hippocratic facies more frequently in this than in any other disease—*"a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the color of the whole face being brown, black, livid, or lead-colored."* There are one or two additional points about the abdomen. The tympany is usually excessive, owing to the great relaxation of the walls of the intestines by inflammation and exudation. The splenic dulness may be obliterated, the diaphragm pushed up, and the apex beat of the heart dislocated to the fourth interspace. The liver dulness may be greatly reduced, or may, in the mammary line, be obliterated. It has been claimed that this is a distinctive feature of perforative peritonitis, but the liver dulness in the middle mammary line may be obliterated by tympanites alone. In the axillary line, on the other hand, the liver dulness, though diminished, may persist. Pneumo-peritoneum following perforation more certainly obliterates the hepatic dulness. In such cases the fluid effused produces a dulness in the lateral region; but with gas in the peritoneum, if the patient is turned on the left side, a clear note is heard beneath the seventh and eighth rib. Acute peritonitis may present a flat, rigid abdomen throughout its course.

Effusion of fluid—ascites—is usually present except in some acute, rapidly fatal cases. The flanks are dull on percussion. The dulness may be movable, though this depends altogether upon the degree of adhesions. There may be considerable effusion without either movable dulness or fluctuation. A friction rub may be present, as first pointed out by Bright, but it is not nearly so common in acute as in chronic peritonitis.

Course.—The acute diffuse peritonitis usually terminates in death. The most intense forms may kill within thirty-six to forty-eight hours; more commonly death results in four or five days, or the attack may be prolonged to eight or ten days. The pulse becomes irregular, the heart-sounds weak, the breathing shallow; there are lividity with pallor, a cold skin with high rectal temperature—a group of symptoms indicating profound failure of the vital functions for which Gee has revived the old term *lipothymia*. Occasionally death occurs with great suddenness, owing, possibly, to paralysis of the heart.

Diagnosis.—In typical cases the severe pain at onset, the distention of the abdomen, the tenderness, the fever, the gradual onset of effusion, collapse, and the vomiting give a characteristic picture. Careful inquiries should at once be made concerning the previous condition, from which a clew can often be had as to the starting-point of the trouble. In young adults a considerable proportion of all cases depends upon perforating appendicitis, and there may be an account of previous attacks of pain in the iliac region, or of constipation alternating with diarrhœa. In women the most frequent causes are suppurative processes in the pelvic viscera, associated with salpingitis, abscesses

Weak heart sounds of the head, usually absence of sounds of pericardium.

in the broad ligaments, or acute puerperal infection. Perforation of gastric ulcer is a more common factor in women than in men. It is not always easy to determine the cause. Many cases come under observation for the first time with the abdomen distended and tender, and it is impossible to make a satisfactory examination. In such instances the pelvic organs should be examined with the greatest care. In typhoid fever, if the patient is conscious, the sudden onset of pain, the tenderness, rigidity, muscle spasm, and the aggravation of the general symptoms indicate what has happened. When the patient is in deep coma, on the other hand, the perforation may be overlooked. The following conditions are most apt to be mistaken for acute peritonitis:

(*a*) *Acute Entero-colitis.*—Here the pain and distention and the sensitiveness on pressure may be marked. The pain is more colicky in character, the diarrhœa is more frequent, and the collapse is more extreme.

(*b*) *The So-called Hysterical Peritonitis.*—This has deceived the very elect, as almost every feature of genuine peritonitis, even the collapse, may be simulated. The onset may be sudden, with severe pain in the abdomen, tenderness, vomiting, diarrhœa, difficulty in micturition, and the characteristic decubitus. Even the temperature may be elevated. There may be recurrence of the attack. A case has been reported by Bristowe in which four attacks occurred within a year, and it was not until special hysterical symptoms developed that the true nature of the trouble was suspected.

(*c*) *Obstruction of the bowel* ~~as already mentioned~~ may simulate peritonitis, both having pain, vomiting, tympanites, and constipation ~~in common.~~ It may for a couple of days ~~really~~ be impossible to make a diagnosis in the absence of a satisfactory history.

(*d*) *Rupture of an abdominal aneurism or embolism of the superior mesenteric artery* may cause symptoms which simulate peritonitis. In the latter, a sudden onset with severe pain, the collapse symptoms, frequent vomiting, and great distention of the abdomen may be present.

(*e*) ~~I have already referred to the fact that~~ acute hæmorrhagic pancreatitis may be mistaken for peritonitis. Lastly, a ruptured tubal pregnancy may resemble acute peritonitis.

II. PERITONITIS IN INFANTS

Peritonitis may occur in the fetus as a consequence of syphilis, and may lead to constriction of the bowel by fibrous adhesions.

In the new-born a septic peritonitis may extend from an inflamed cord. Distention of the abdomen, slight swelling and redness about the cord, and not infrequently jaundice are present. It is ~~an~~ uncommon ~~event~~, and existed in only 4 of 51 infants dying with inflammation of the cord and septicæmia (Runge).

During childhood peritonitis arises from causes similar to those affecting the adult. Perforative appendicitis is common. Peritonitis following blows or kicks on the abdomen occurs more frequently at this period. In boys injury while playing football may be followed by diffuse peritonitis. A rare cause in children is extension through the diaphragm from an empyema. There are on record instances of peritonitis occurring in several children at the same

school, and it has been attributed to sewer-gas poisoning. It was in investigating an epidemic of this kind at the Wandsworth school, in London, that Anstie received the post mortem wound of which he died. It is to be remembered that peritonitis in children may follow the gonorrhœal vulvitis so common in infant homes and hospitals.

III. LOCALIZED PERITONITIS

Subphrenic Peritonitis.—The general peritoneum covering the right and left lobes of the liver may be involved in an extension from the pleura of suppurative, tuberculous, or cancerous processes. In various affections of the liver—cancer, abscess, hydatid disease, and in affections of the gall-bladder—the inflammation may be localized to the peritoneum covering the upper surface of the organ. These forms of localized subphrenic peritonitis in the greater sac are not so important in reality as those which occur in the lesser peritoneum. The anatomical relations of this structure are as follows: It lies behind and below the stomach, the gastro-hepatic omentum, and the anterior layer of the great omentum. Its lower limit forms the upper layer of the transverse meso-colon. On either side it reaches from the hepatic to the splenic flexure of the colon, and from the foramen of Winslow to the hilus of the spleen. Behind it covers and is tightly adherent to the front of the pancreas. Its upper limit is formed by the transverse fissure of the liver, and by that portion of the diaphragm which is covered by the lower layer of the right lateral ligament of the liver; the lobus Spigelii lies bare in the cavity. The foramen of Winslow, through which the lesser communicates with the greater peritoneum, is readily closed by inflammation.

Inflammatory processes, exudates, and hæmorrhages may be confined entirely to the lesser peritoneum. The exudate of tuberculous peritonitis may be confined to it. Perforations of certain parts of the stomach, of the duodenum, and of the colon may excite inflammation in it alone; and in various affections of the pancreas, particularly trauma and hæmorrhage, the effusion into the sac has often been confounded with cyst of this organ.

Special mention must be made of the remarkable form of subphrenic abscess containing air, which may simulate closely pneumothorax, and hence was called by Leyden *pyo-pneumothorax subphrenicus*. The affection has been thoroughly studied by Scheurlen, Mason, Meltzer, and Lee Dickinson. In 142 out of 170 recorded cases the cause was known. In a few instances, as in one reported by Meltzer, the subphrenic abscess seemed to have followed pneumonia. Pyothorax is an occasional cause. By far the most frequent condition is gastric ulcer, which occurred in 80 of the cases. Duodenal ulcer was the cause in 6 per cent. In about 10 per cent. of the cases the appendix was the starting-point of the abscess. Cancer of the stomach is an occasional cause. Other rare causes are trauma, which was present in one of my cases, perforation of an hepatic or a renal abscess, lesions of the spleen, abscess, and cysts of the pancreas.

In a majority of all the cases in which the stomach or duodenum is perforated—sometimes, indeed, in the cases following trauma, as in *Case 3* of my series—the abscess contains air.

The symptoms of subphrenic abscess vary very considerably, depending a good deal upon the primary cause. The onset, as a rule, is abrupt, particularly when due to perforation of a gastric ulcer. There are severe pain, vomiting, often of bilious or of bloody material; respiration is embarrassed, owing to the involvement of the diaphragm; then the constitutional symptoms occur associated with suppuration, chills, irregular fever, and emaciation. Subsequently perforation may take place into the pleura or into the lung, with severe cough and abundant purulent expectoration.

The perihepatic abscess beneath the arch of the diaphragm, whether to the right or left of the suspensory ligament, when it does not contain air, is almost invariably mistaken for empyema. Remarkable features are superadded when the abscess cavity contains air. On the right side, when the abscess is in the greater peritoneum, above the right lobe of the liver, the diaphragm may be pushed up to the level of the second or third rib, and the physical signs on percussion and auscultation are those of pneumothorax, particularly the tympanitic resonance and the movable dulness. The liver is usually greatly depressed and there is bulging on the right side. Still more obscure are the cases of air-containing abscesses due to perforation of the stomach or duodenum, in which the gas is contained in the lesser peritoneum. Here the diaphragm is pushed up and there are signs of pneumothorax on the left side. In a large majority of all the cases which follow perforation of a gastric ulcer the effusion lies between the diaphragm above, and the spleen, stomach, and the left lobe of the liver below.

The prognosis in subphrenic abscess is not very hopeful. Of the cases on record about 20 per cent. only have recovered.

Appendicular.—The most frequent cause of localized peritonitis in the male is inflammation of the appendix vermiformis. The situation varies with the position of this extremely variable organ. The adhesion, perforation, and intraperitoneal abscess cavity may be within the pelvis, or to the left of the median line in the iliac region, in the lower right quadrant of the umbilical region—a not uncommon situation—or, of course, most frequently in the right iliac fossa. In the most common situation the localized abscess lies upon the psoas muscle, bounded by the cæcum on the right and the terminal portion of the ileum and its mesentery in front and to the left. In many of these cases the limitation is perfect, and post mortem records show that complete healing may take place with the obliteration of the appendix in a mass of firm scar tissue.

Pelvic Peritonitis.—The most frequent cause is inflammation about the uterus and Fallopian tubes. Puerperal septicæmia, gonorrhœa, and tuberculosis are the usual causes. The tubes are the starting-point in a majority of the cases. The fimbriæ become adherent and closely matted to the ovary, and there is gradually produced a condition of thickening of the parts, in which the individual organs are scarcely recognizable. The tubes are dilated and filled with cheesy matter or pus, and there may be small abscess cavities in the broad ligaments. Rupture of one of these may cause general peritonitis, or the membrane may be involved by extension, as in tuberculosis of these parts.

IV. CHRONIC PERITONITIS

The following varieties may be recognized:

Local adhesive peritonitis, a very common condition, which occurs particularly about the spleen, forming adhesions between the capsule and the diaphragm, about the liver, less frequently about the intestines and mesentery. Points of thickening or puckering on the peritoneum occur sometimes with union of the coils or with fibrous bands. In a majority of such cases the condition is met accidentally post mortem. Two sets of symptoms may, however, be caused by these adhesions. When a fibrous band is attached in such a way as to form a loop or snare, a coil of intestine may pass through it. Thus, of the 295 cases of intestinal obstruction analyzed by Fitz, 63 were due to this cause. The second group is less serious and comprises cases with persistent abdominal pain of a colicky character, sometimes rendering life miserable.

Diffuse Adhesive Peritonitis.—This is a consequence of an acute inflammation, either simple or tuberculous. The peritoneum is obliterated. On cutting through the abdominal wall, the coils of intestines are uniformly matted together and can neither be separated from each other nor can the visceral and parietal layers be distinguished. There may be thickening of the layers, and the liver and spleen are usually involved in the adhesions.

Proliferative Peritonitis.—Apart from cancer and tubercle, which produce typical lesions of chronic peritonitis, the most characteristic form is that which may be described under this heading. The essential anatomical feature is great thickening of the peritoneal layers, usually without much adhesion. The cases are sometimes seen with sclerosis of the stomach. In one instance I found it in connection with a sclerotic condition of the cæcum and the first part of the colon. It is not uncommon with cirrhosis of the liver. In ~~the inspection of~~ a case of this kind there is usually moderate effusion, more rarely extensive ascites. The peritoneum is opaque white in color, and everywhere thickened, often in patches. The omentum is usually rolled and forms a thickened mass transversely placed between the stomach and the colon. The peritoneum over the stomach, intestines, and mesentery is sometimes greatly thickened. The liver and spleen may simply be adherent, or there is a condition of chronic perihepatitis or perisplenitis, so that a layer of firm, almost gristly connective tissue of from one-fourth to half an inch in thickness encircles these organs. Usually the volume of the liver is in consequence greatly reduced. The gastro-hepatic omentum may be constricted by this new growth and the calibre of the portal vein much narrowed. A serous effusion may be present. On account of the adhesions which form, the peritoneum may be divided into three or four different sacs, as is ~~more fully~~ described under ~~the~~ tuberculous peritonitis. In these cases the intestines are usually free, though the mesentery is greatly shortened. There are instances of chronic peritonitis in which the mesentery is so shortened by this proliferative change that the intestines form a ball not larger than a cocoanut situated in the middle line, and after the removal of the exudation can be felt as a solid tumor. The intestinal wall is greatly thickened and the mucous membrane of the ileum is thrown into folds like the valvulæ conniventes. This proliferative peritonitis is found frequently in the subjects of chronic alcoholism. In cases of

long-continued ascites the serous surfaces generally become thickened and present an opaque, dead white color. This condition is observed especially in hepatic cirrhosis, but attends tumors, chronic passive congestion, etc.

In all forms of chronic peritonitis a friction may be felt usually in the upper zone of the abdomen. Polyorrhomenitis, polyserositis, general chronic inflammation of the serous membranes, Concato's disease (as the Italians call it) may occur with this form as well as in the tuberculous variety. The pericardium and both pleuræ may be involved.

In some instances of chronic peritonitis the membrane presents numerous nodular thickenings, which may be mistaken for tubercles. J. F. Payne described a case of this sort associated with disseminated growths throughout the liver which were not cancerous. It has been suggested that some of the cases of tuberculous peritonitis cured by operation have been of this nature, but histological examination would, as a rule, readily determine between the conditions. Miura, in Japan, has reported a case in which these nodules contained the ova of a parasite. One case has been reported in which the exciting cause was regarded as cholesterin plates, which were contained within the granulomatous nodules.

Chronic Hæmorrhagic Peritonitis.—Blood-stained effusions in the peritoneum occur particularly in cancerous and tuberculous disease. A chronic inflammation analogous to the hæmorrhagic pachymeningitis of the brain was described first by Virchow, and is localized most commonly in the pelvis. Layers of new connective tissue form on the surface of the peritoneum with large wide vessels from which hæmorrhage occurs. This is repeated from time to time with the formation of regular layers of hæmorrhagic effusion. It is rarely diffuse, more commonly circumscribed. Probably the spontaneous peritoneal hæmorrhage with the features of an "acute abdomen" (Churchman) may represent the primary form of this rare condition.

V. NEW GROWTHS IN THE PERITONEUM

Tuberculous Peritonitis.—This has ~~already~~ been considered.

Cancer of the Peritoneum.—Although, as a rule, secondary to disease of the stomach, liver, or pelvic organs, cases of primary cancer have been described. It is probable that the so-called primary cancers of the serous membranes are endotheliomata and not carcinomata. Secondary malignant peritonitis occurs in connection with all forms of cancer. It is usually characterized by a number of round tumors scattered over the entire peritoneum, sometimes small and miliary, at other times large and nodular, with puckered centres. The disease most commonly starts from the stomach or the ovaries. The omentum is indurated and, as in tuberculous peritonitis, forms a mass which lies transversely across the upper portion of the abdomen. Primary malignant disease of the peritoneum is extremely rare. Colloid is said to have occurred, forming enormous masses, which in one case weighed over 100 pounds. Cancer of this membrane spreads, either by the detachment of small particles which are carried in the lymph currents and by the movements to distant parts, or by contact of opposing surfaces. It occurs more frequently in women than in men, and more commonly at the later period of life.

The *diagnosis* of cancer of the peritoneum is easy with a history of a local malignant disease; as when it occurs with ovarian tumor or with cancer of the pylorus. In cases in which there is no evidence of a primary lesion the diagnosis may be doubtful. The clinical picture is usually that of chronic ascites with progressive emaciation. There may be no fever. If there is much effusion nothing definite can be felt on examination. After tapping, irregular nodules or the curled omentum may be felt lying transversely across the upper portion of the abdomen. Multiple nodules, if large, indicate cancer, particularly in persons above middle life. Nodular tuberculous peritonitis is most frequent in children. The presence about the navel of secondary nodules and indurated masses is more common in cancer. Inflammation, suppuration, and the discharge of pus from the navel rarely occur except in tuberculous disease. Considerable enlargement of the inguinal glands may be present in cancer. The nature of the fluid in cancer and in tubercle may be much alike. It may be hæmorrhagic in both; more often in the latter. The histological examination in cancer may show large multinuclear cells or groups of cells—the sprouting cell-groups of Foulis—which are extremely suggestive. The colloid cancer may produce a totally different picture; instead of ascitic fluid, the abdomen is occupied by the semi-solid gelatinous substance, and is firm, not fluctuating.

And, lastly, there are instances of echinococci in the peritoneum which may simulate cancer very closely.

VI. ASCITES

(*Hydro-peritoneum*)

Definition.—The accumulation of serous fluid in the peritoneal cavity.

Etiology.—Local Causes.—(*a*) Chronic inflammation of the peritoneum, either simple, cancerous, or tuberculous. (*b*) Portal obstruction in the terminal branches within the liver, as in cirrhosis and chronic passive congestion, or by compression of the vein in the gastro-hepatic omentum, either by proliferative peritonitis, by new growths, or by aneurism. (*c*) Thrombosis of the portal vein. (*d*) Tumors of the abdomen. The solid growths of the ovaries may cause considerable ascites, which may completely mask the true condition. It is important to bear in mind this possibility in the obscure ascites of women. The condition is not infrequent, as I saw four cases due to this cause in 1911. The enlarged spleen in leukæmia, less commonly in malaria, may be associated with recurring ascites.

General Causes.—The ascites is part of a general dropsy, the result of mechanical effects, as in heart-disease, chronic emphysema, and sclerosis of the lung. In cardiac lesions the effusion is sometimes confined to the peritoneum, in which case it is due to secondary changes in the liver, or it has been suggested to be connected with a failure of the suction action of this organ by which the peritoneum is kept dry. Ascites occurs also in the dropsy of Bright's disease, and in hydræmic states of the blood.

Symptoms.—A gradual uniform enlargement of the abdomen is the char-

acteristic symptom of ascites. The PHYSICAL SIGNS are usually distinctive. (*a*) *Inspection.*—According to the amount of fluid the abdomen is protuberant and flattened at the sides. With large effusions, the skin is tense and may present the lineæ albicantes. Frequently the navel itself and the parts about it are very prominent. In many cases the superficial veins are enlarged and a plexus joining the mammary vessels can be seen. Sometimes it can be determined by pressure on these veins that the current is from below upward. In some instances, as in thrombosis or obliteration of the portal vein, these superficial abdominal vessels may be extensively varicose. About the navel in cases of cirrhosis there is occasionally a large bunch of distended veins, the so-called caput Medusæ. The heart may be displaced upward.

(*b*) *Palpation.*—Fluctuation is obtained by placing the fingers of one hand upon one side of the abdomen and by giving a sharp tap on the opposite side with the other hand, when a wave is felt to strike as a definite shock against the applied fingers. Even comparatively small quantities of fluid may give this fluctuation shock. When the abdominal walls are thick or very fat, an assistant may place the edge of the hand ~~or a piece of cardboard~~ in front of the abdomen. A different procedure is adopted in palpating for the solid organs in case of ascites. Instead of placing the hand flat upon the abdomen, as in the ordinary method, the pads of the fingers only are placed lightly upon the skin, and then by a sudden depression of the fingers the fluid is displaced and the solid organ or tumor may be felt. By this method of "dipping" or displacement, ~~as it is called,~~ the liver may be felt below the costal margin, or the spleen, or sometimes solid tumors of the omentum or intestine.

(*c*) *Percussion.*—In the dorsal position with a moderate quantity of fluid in the peritoneum the flanks are dull, while the umbilical and epigastric regions, in which the intestines float, are tympanitic. This area of clear resonance may have an oval outline. Having obtained the lateral limit of the dulness on one side, if the patient turns on the opposite side, the fluid gravitates to the dependent part and the uppermost flank is now tympanitic. In moderate effusions this movable dulness changes greatly in the different postures. Small amounts of fluid, probably under a litre, would scarcely give movable dulness, as the pelvis and the renal regions hold a considerable quantity. In such cases it is best to place the patient in the knee-elbow position, when a dull note will be determined at the most dependent portion. By careful attention to these details mistakes are usually avoided.

Differential Diagnosis.—The following are among the conditions which may be mistaken for dropsy: *Ovarian tumor,* in which the sac develops, as a rule, unilaterally, though when large it is centrally placed. The dulness is anterior and the resonance is in the flanks, into which the intestines are pushed by the cyst. Examination *per vaginam* may give important indications. In those rare instances in which gas develops in the cyst the diagnosis may be very difficult. Succussion has been obtained in such cases. A *distended bladder* may reach above the umbilicus. In such instances some urine dribbles away, and suspicion of ascites or a cyst is occasionally entertained. I once saw a trocar thrust into a distended bladder, which was supposed to be an ovarian cyst, and it is stated that John Hunter tapped a bladder, supposing it to be ascites. Such a mistake should be avoided by careful catheterization

prior to any operative procedures. And lastly, there are large pancreatic or hydatid cysts in the abdomen which may simulate ascites.

Nature of the Ascitic Fluid.—Usually this is a clear serum, light yellow in the ascites of anæmia and Bright's disease, often darker in color in cirrhosis of the liver. The specific gravity is low, seldom more than 1.010 or 1.015, whereas in the fluid of ovarian cysts or chronic peritonitis the specific gravity is over 1.015. It is albuminous and sometimes coagulates spontaneously. Dock has called attention to the importance of the study of the cells in the exudate. In cancer very characteristic forms, with nuclear figures, may be found. Hæmorrhagic effusion usually occurs in cancer and tuberculosis, and occasionally in cirrhosis. I have already referred to the instances of hæmorrhagic effusion in connection with ruptured tubal pregnancy.

Chylous Ascites.—Of the cases tabulated by MacKenzie, Wallis, and Scholberg, 81 were in association with tumors, 16 with the infections, chiefly tuberculosis, 37 in association with affections of the thoracic duct and lymphatic system, and 78 in connection with general diseases such as cirrhosis of the liver, cardiac disease, nephritis, amyloid disease, and thrombosis of the blood-vessels. In a certain number of cases the cause of the condition is unknown. Quincke recognized that there were two types, one in which there was a true milky or fatty fluid, the other in which the turbidity is due to fatty degeneration of cells or to chemical substances of a non-fatty nature. The fluid of the true chylous ascites is yellowish-white in color, contains fine fat globules, a creamy layer collects on standing, the specific gravity generally exceeds 1.012, and the fat content is high. As a rule, it tends to accumulate rapidly and large amounts may be removed. The fluid of pseudo-chylous ascites is milky white, the opacity often may vary at different tappings. Microscopically there are many fine refractile granules, but they do not give reactions for fat, the cellular elements may be numerous, and a creamy layer rarely forms. The specific gravity is less than 1.012, and the total solids rarely exceed 2 per cent. The fat content is low. Lecithin combined with globulin appears to be the cause of the opalescence. The authors from whom I have quoted conclude that milky ascites is characteristic of no specific morbid lesion. The prognosis is usually grave.

Treatment of the Previous Conditions.—(*a*) Acute Peritonitis.—Rest is enjoined upon the patient by the severe pain which follows the slightest movement, and he should be propped in the position which gives him greatest relief. Whether morphia should be given will depend upon the cause. In the pain of appendicitis and of perforation in typhoid fever it is best to use an ice-bag and withhold the drug. Late in the disease and in hopeless conditions it may be given freely. The opium treatment so strongly advocated by the late Alonzo Clark has gone out of vogue.

Local applications—the ice-bag, hot turpentine stupes, or cloths wrung out of ice-water—may be laid upon the abdomen.

The question of the use of purgatives in peritonitis has of late been warmly discussed. Theoretically it appears correct to give salines in concentrated form, which cause a rapid and profuse exosmosis of serum from the intestinal vessels, relieving the congestion and reducing the œdema, which is one important factor in causing the meteorism. It is also urged that the increased peristalsis prevents the formation of adhesions. In reading the reports of

these successful cases, one is not always convinced, however, that peritonitis actually existed. Still, in cases of acute peritonitis due to extension or following operation or in septic conditions the judgment of many careful men is decidedly in favor of the use of salines. The majority of cases of peritonitis which come under the care of the physician follow lesions of the abdominal viscera or are due to perforation of ulcer of the stomach, the ileum, or the appendix. In such cases, particularly in the large group of appendix cases, to give saline purgatives is, to say the least, most injudicious treatment. In these instances rectal injections should be employed to relieve the large bowel. No symptom in acute peritonitis is more serious than the tympanites, and none is more difficult to meet. The use of the long tube and injections containing turpentine may be tried. Drugs by the mouth can not be retained.

For the vomiting, ice and small quantities of soda water may be employed. The patient should be fed on milk, but if the vomiting is distressing it is best not to attempt to give food by the mouth, but to use small nutrient enemata. In all cases it is best to have a surgeon in consultation early in the disease, as the question of operation may come up at any moment. In the acute forms of tuberculous peritonitis operative measures appear to be more hopeful, but they are not always successful.

(*b*) Chronic Peritonitis.—For the cases of chronic proliferative peritonitis very little can be done. The treatment is practically that of ascites. In all these forms, when the distention becomes extreme, tapping is indicated. The treatment of tuberculous peritonitis has fallen largely into the hands of the surgeons, but the results depend on the stage at which the operation is performed and the variety of the disease. With ascites the outlook is good; but when there are tuberculous tumors and many adhesions the results are not very satisfactory. Maurice Richardson, in a child aged five, with a suspected appendicitis (tumor, etc.), found the symptoms to be due to enlarged, tuberculous mesenteric glands, which were removed, and the boy remained well five years after the operation.

(*c*) Ascites.—The treatment depends somewhat on the nature of the case. In cirrhosis early and repeated tapping may give time for the establishment of the collateral circulation, and temporary cures have followed this procedure. Permanent drainage with Southey's tube, incision, and washing out the peritoneum have also been practiced. In the ascites of cardiac and renal disease the cathartics are most satisfactory, particularly the bitartrate of potash, given alone or with jalap, and the large doses of salts given an hour before breakfast with as little water as possible. These sometimes cause rapid disappearance of the effusion, but they are not so successful in ascites as in pleurisy with effusion. The stronger cathartics may sometimes be necessary. The ascites forming part of the general anasarca of Bright's disease will receive consideration under another section.

SECTION VI

DISEASES OF THE RESPIRATORY SYSTEM

A. DISEASES OF THE NOSE

I. AUTUMNAL CATARRH

(*Hay Fever*)

Definition.—An affection of the upper air-passages, often associated with asthmatic attacks, due to the action of the pollen of certain grasses and plants upon a hypersensitive mucous membrane.

Etiology.—This affection was first described in 1819 by Bostock, who called it *catarrhus æstivus*. Morrill Wyman, of Cambridge, Mass., wrote a monograph on the subject, and described two forms, the "June cold," or "rose cold," which comes on in the spring, and the autumnal form, which, in the United States, comes on in August and September, and never persists after a severe frost. In the Southern States cases occur all through the year. It is more common in America and in Great Britain than on the Continent. The disposition to the disease is hereditary. Women are more subject to it than men. Young and middle-aged persons are most often attacked. The tendency lessens as age advances. Dwellers in cities are chiefly attacked. The educated and highly nervous are most susceptible. The disease affects certain families, and Beard found an hereditary factor in 33 per cent. of his cases. A morbid sensitiveness of the nasal mucosa is present in many cases.

The disease must be differentiated from nervous coryza (which has been induced by suggestion) and from the attacks of irritation of the nasal, conjunctival, and bronchial mucous membranes excited by the odor of a horse, or of the "harmless necessary cat."

Dunbar's researches have placed the etiology of the disease on a scientific basis. He has shown that there is but one cause, the pollen of grasses and certain plants. The pollen of about 130 different plants has now been examined, of which that of 25 grasses and of only 7 other kinds of plants exert a definite action. The pollen of rye is the most active. Dunbar and his students have found that the severity of hay-fever attacks is in direct proportion to the quantity of pollen present in the atmosphere. In persons predisposed to the disease the pollen applied to the conjunctivæ or nasal mucosa excites characteristic attacks. He has isolated a peculiar poison of an albuminous nature from the pollen. It is so powerful that .000025 milligram excites irritation in the conjunctiva of a susceptible subject. This is the amount of toxin which corresponds to two or three pollen grains. It is entirely without

Def. The mec. mech. of those susceptible

anaphylactic skin reaction by epidermal of pollen. Some react only to 1 pollen others to several varieties

active immunization by pollen epidermal mucosal

anaphylactic skin reaction given to pollen. (vaccine)

The pollens are [illegible]

The object of this pollen is direct on the m.m. by irritation and indirect by absorption of toxalbumin.

Sed hicarb. Zi Dres hymneth "

Sometimes also nasal spray.

acidosis bound in serum

Active immunization by pollen extracts is sometimes [illegible] effective.

Epilepsy in 1915 - [illegible]

influence on normal persons. In larger doses severer attacks are caused, and, injected subcutaneously, it has been followed by very unpleasant symptoms. He has succeeded in obtaining an antitoxin by injecting the poison into animals. It is capable of cutting short attacks of ordinary hay fever.

Symptoms.—These are, in a majority of the cases, very like those of ordinary coryza. There may, however, be much more headache and distress, and some patients become very low-spirited. At the outset, or even daily throughout the attack, sneezing may be frequent. Cough is a common symptom and may be very distressing. Paroxysms of asthma may occur indistinguishable from the ordinary bronchial form. The two conditions may indeed alternate, the patient having at one time an attack of common hay fever and at another, under similar circumstances, an attack of bronchial asthma.

Treatment.—This may be comprised under four heads: First, remedies may be given to improve the stability of the nervous system—such as arsenic, phosphorus, and strychnia. Secondly, climatic. Dwellers in the cities of the Atlantic seaboard and of the Central States enjoy complete immunity in the Adirondacks and White Mountains. As a rule the disease is aggravated by residence in agricultural districts. The dry mountain air is unquestionably the best; there is no general rule, and there are cases which do well at the seaside. Thirdly, the thorough local treatment of the nose, particularly the destruction of the vessels and sinuses over the sensitive areas. Fourthly, the antitoxin treatment of Dunbar in suitable cases gives excellent results when used as a prophylactic. Owing to the peculiar nature of the disease and the constant reinfection of the mucous membranes by pollen on exposure to the outside air, it is advised to sleep with the windows closed and to apply the serum in the morning before rising both to eyes and nose, and again during the day if ~~the slightest~~ irritation is felt in the conjunctivæ or nasal mucous membranes. ~~The *Pollantin* of Dunbar is now "on the market."~~

II. EPISTAXIS

Etiology.—Bleeding from the nose may result from local or constitutional conditions. Among local causes may be mentioned traumatism, small ulcers, picking or scratching the nose, new growths, and the presence of foreign bodies. In chronic nasal catarrh bleeding is not infrequent. The blood may come from one or both nostrils. The flow may be profuse after an injury.

Among general conditions with which nose-bleeding is associated, the following are the most important: It occurs in growing children, particularly about the age of puberty; more frequently in the delicate than in the strong and vigorous. There is a family form in which many members in several generations are affected. I have described a remarkable hereditary multiple telangiectasis, a special feature of which is recurring epistaxis. The disease has nothing to do with hæmophilia, with which it has been confounded. The bleeding occurs from the telangiectasis in the nasal mucosa, and from those in the lips, tongue, and skin. A severe anæmia may be caused by the loss of blood.

Epistaxis is a very common event in persons of so-called plethoric habit. It is stated sometimes to precede, or to indicate a liability to, apoplexy. In

venous engorgement epistaxis is not common and there may be a most extreme grade of cyanosis without its occurrence. It is frequent in cirrhosis hepatis. In balloon and mountain ascensions, in the very rarefied atmosphere, hæmorrhage from the nose is a common event. In hæmophilia the nose ranks first of the mucous membranes from which bleeding arises. It occurs in all forms of chronic anæmias, in chronic interstitial nephritis, and in cirrhosis of the liver. It precedes the onset of certain fevers, more particularly typhoid, with which it seems associated in a special manner. Vicarious epistaxis has been described in cases of suppression of the menses. Lastly, it is said to be brought on by certain psychical impressions, but the observations on this point are not trustworthy. The blood in epistaxis results from capillary oozing or diapedesis. The mucous membrane is deeply congested and there are often capillary angiomata situated usually in the respiratory portion of the nostril and upon the cartilaginous septum.

Symptoms.—Slight hæmorrhage is not associated with any special features. When the bleeding is protracted the patients have the more serious manifestations of loss of blood. In the slow dripping which takes place in some instances of hæmophilia, there may be formed a remarkable blood tumor projecting from one nostril and extending even below the mouth.

Death from ordinary epistaxis is very rare. The more blood is lost the greater is the tendency to clotting with spontaneous cessation of the bleeding.

Diagnosis.—The diagnosis is usually easy. One point only need be mentioned; namely, that bleeding from the posterior nares occasionally occurs during sleep and the blood trickles into the pharynx and may be swallowed. If vomited, it may be confounded with hæmatemesis; or, if coughed up, with hæmoptysis.

Treatment.—In a majority of the cases the bleeding ceases of itself. Various simple measures may be employed, such as holding the arms above the head, the application of ice to the nose, or the injection of cold or hot water into the nostrils. Astringents, such as zinc, alum, or tannin, may be used; and the tincture of the perchloride of iron, diluted with ice-water, may be introduced into the nostrils. If the bleeding comes from an ulcerated surface, an attempt should be made to apply chromic acid or the cautery. If the bleeding is at all severe and obstinate, the posterior nares should be plugged. One of the patients with epistaxis and spider angiomata of the skin and mucous membranes used a finger of a rubber glove with a small rubber tube and stopcock by which he could dilate the glove finger, inserted into the nostril, and so effectually control the bleeding. The inhalation of carbonic-acid gas may be tried or a solution of gelatine or of adrenalin injected into the nostril.

B. DISEASES OF THE LARYNX

I. ACUTE CATARRHAL LARYNGITIS

This may come on as an independent affection or in association with general catarrh of the upper respiratory passages.

Etiology.—Many cases are due to catching cold or to overuse of the voice;

Pneumococcus, influenza bac. e Mic. cat. anche
durante le organismi.

others come on in consequence of the inhalation of irritating gases. It may occur in the general catarrh associated with influenza and measles. Very severe laryngitis is excited by traumatism, either injuries from without or the lodgment of foreign bodies. It may be caused by the action of very hot liquids or corrosive poisons.

Symptoms.—There is a sense of tickling referred to the larynx; the cold air irritates and, owing to the increased sensibility of the mucous membrane, the act of inspiration may be painful. There is a dry cough, and the voice is altered. At first it is simply husky, but soon phonation becomes painful, and finally the voice may be completely lost. In adults the respirations are not increased in frequency, but in children dyspnœa is not uncommon and may occur in spasmodic attacks and become urgent if there is much œdema with the inflammatory swelling.

The laryngoscope shows a swollen mucous membrane of the larynx, particularly the ary-epiglottidean folds. The vocal cords have lost their smooth and shining appearance and are reddened and swollen. Their mobility also is greatly impaired, owing to the infiltration of the adjoining mucous membrane and of the muscles. A slight mucoid exudation covers the parts. The constitutional symptoms are not severe. There is rarely much fever, and in many cases the patient is not seriously ill. Occasionally cases come on with greater intensity, the cough is very distressing, deglutition is painful, and there may be urgent dyspnœa.

Diagnosis.—There is rarely any difficulty in determining the nature of a case if a satisfactory laryngoscopic examination can be made. The severer forms may simulate œdema of the glottis. When the loss of voice is marked, the case may be mistaken for one of nervous aphonia, but the laryngoscope would decide the question at once. Much more difficult is the diagnosis of acute laryngitis in children, particularly in the very young, in whom it is so hard to make a proper examination. From ordinary laryngismus it is to be distinguished by the presence of fever, the mode of onset, and particularly the coryza and the previous symptoms of hoarseness or loss of voice. Membranous laryngitis may at first be quite impossible to differentiate, but in a majority of cases of this affection there are patches on the pharynx and early swelling of the cervical glands. The symptoms, too, are much more severe.

Treatment.—Rest of the larynx should be enjoined, so far as phonation is concerned; smoking should be forbidden. In cases of any severity the patient should be kept in bed. The room should be at an even temperature and the air saturated with moisture. Early in the disease, if there is much fever, aconite and citrate of potash may be given, and for the irritating painful cough a full dose of Dover's powder at night. An ice-bag externally often gives great relief.

II. CHRONIC LARYNGITIS

Etiology.—The cases usually follow repeated acute attacks. The most common cause is overuse of the voice, particularly in persons whose occupation necessitates shouting in the open air. The constant inhalation of irritating substances, as tobacco-smoke, may also cause it.

Symptoms.—The voice is usually hoarse and rough and in severe cases may

be almost lost. There is usually very little pain; only the unpleasant sense of tickling in the larynx, which causes a frequent desire to cough. With the laryngoscope the mucous membrane looks swollen, but much less red than in the acute condition. In association with the granular pharyngitis, the mucous glands of the epiglottis and of the ventricles may be involved.

Treatment.—The nostrils should be carefully examined, since in some instances chronic laryngitis is associated with and even dependent upon obstruction to the free passage of air through the nose. Local application must be made directly to the larynx, either with a brush or by means of a spray. Among the remedies most recommended are the solutions of nitrate of silver, chlorate of potash, perchloride of zinc, and tannic acid. Insufflations of bismuth are sometimes useful.

Among directions to be given are the avoidance of heated rooms and loud speaking, and abstinence from tobacco and alcohol. The throat should not be too much muffled, and morning and evening the neck should be sponged with cold water.

III. ŒDEMATOUS LARYNGITIS

Etiology.—Œdema of the glottis, or, more correctly, of the structures which form the glottis, a very serious affection, is met with (*a*) as a rare sequence of ordinary acute laryngitis; (*b*) in chronic diseases of the larynx, as syphilis or tubercle; (*c*) in severe inflammatory diseases like diphtheria, in erysipelas of the neck, and in various forms of cellulitis; (*d*) occasionally in the acute infectious diseases—scarlet fever, typhus, or typhoid; in Bright's disease, either acute or chronic, there may be a rapidly developing œdema; (*e*) in angio-neurotic œdema.

Symptoms.—There is dyspnœa, increasing in intensity, so that within an hour or two the condition becomes very critical. There is sometimes marked stridor in respiration. The voice becomes husky and disappears. The laryngoscope shows enormous swelling of the epiglottis, which can sometimes be felt with the finger or even seen when the tongue is strongly depressed with a spatula. The ary-epiglottidean folds are the seat of the chief swelling and may almost meet in the middle line. Occasionally the œdema is below the true cords.

The diagnosis is rarely difficult, inasmuch as even without the laryngoscope the swollen epiglottis can be seen or felt with the finger. The condition is very often fatal.

Treatment.—An ice-bag should be placed on the larynx, and the patient given ice to suck. The air of the room should be moist. If the symptoms are urgent, the throat should be sprayed with a strong solution of cocaine or adrenalin and the swollen epiglottis scarified. If relief does not follow, tracheotomy should immediately be performed. The high rate of mortality is due to the fact that this operation is, as a rule, too long delayed.

IV. SPASMODIC LARYNGITIS

(*Laryngismus stridulus*)

Spasm of the glottis is met with in many affections of the larynx, but there is a special disease in children which has received the above-mentioned and other names.

Etiology.—A purely nervous affection, without any inflammatory condition of the larynx, it occurs in children between the ages of six months and three years, and is most commonly seen in connection with rickets. As Escherich has shown, the disease has close relations with tetany and may display many of the accessory phenomena of this disease. Often the attack comes on when the child has been crossed or scolded. Mothers sometimes call the attacks "passion fits" or attacks of "holding the breath." It was supposed at one time that they were associated with enlargement of the thymus, and the condition therefore received the name of *thymic asthma*.

The actual state of the larynx during a paroxysm is a spasm of the adductors, but the precise nature of the influences causing it is not yet known, whether centric or reflex from peripheral irritation. The disease is not so common in America as in England.

Symptoms.—The attacks may come on either in the night or in the day; often just as the child awakes. There is no cough, no hoarseness, but the respiration is arrested and the child struggles for breath, the face gets congested, and then, with a sudden relaxation of the spasm, the air is drawn into the lungs with a high-pitched crowing sound, which has given to the affection the name of "child-crowing." Convulsions may occur during an attack or there may be carpo-pedal spasms. Death may, but rarely does, occur during the attack. With the cyanosis the spasm relaxes and respiration begins. The attacks may recur with great frequency throughout the day.

Treatment.—The gums should be carefully examined and, if swollen and hot, freely lanced. The bowels should be carefully regulated and, as these children are usually delicate or rickety, nourishing diet and cod-liver oil should be given. By far the most satisfactory method of treatment is the cold sponging. In severe cases, two or three times a day the child should be placed in a warm bath, and the back and chest thoroughly sponged for a minute or two with cold water. Since learning this practice from Ringer, at the University Hospital, London, I have seen many cases in which it proved successful. It may be employed when the child is in a paroxysm, though if the attack is severe and the lividity is great it is much better to dash cold water into the face. Sometimes the introduction of the finger far back into the throat will relieve the spasm.

Spasmodic croup, believed to be a functional spasm of the muscles of the larynx, is an affection seen most commonly between the ages of two and five years. According to Trousseau's description, the child goes to bed well, and about midnight or in the early morning hours awakes with oppressed breathing, harsh, croupy cough, and perhaps some huskiness of voice. The oppression and distress for a time are very serious, the face is congested, and there are signs of approaching cyanosis. The attack passes off abruptly, the child

falls asleep and awakes the next morning feeling perfectly well. These attacks may be repeated for several nights in succession, and usually cause great alarm to the parents. Whether this is entirely a functional spasm is, I think, doubtful. There are instances in which the child is somewhat hoarse throughout the day, and has slight catarrhal symptoms and a brazen, croupy cough. There is probably slight catarrhal laryngitis with it. These cases are not infrequently mistaken for true croup, and parents are sometimes unnecessarily disturbed by the serious view which the physician takes of the case. Too often the poor child, deluged with drugs, is longer in recovering from the treatment than he would be from the disease. To allay the spasm a whiff of chloroform may be administered, which will in a few moments give relief, or the child may be placed in a hot bath. A prompt emetic, such as wine of ipecac, will usually relieve the spasm, and is specially indicated if the child has overloaded the stomach through the day.

V. TUBERCULOUS LARYNGITIS

Etiology.—Tubercles may arise primarily in the laryngeal mucosa, but in the great majority of cases the affection is secondary to pulmonary tuberculosis, in which it is met with in a variable proportion of from 18 to 30 per cent. Laryngitis may occur very early in pulmonary tuberculosis. There may be well-marked involvement of the larynx with signs of very limited trouble at one apex. These are cases which, in my experience, run a very unfavorable course.

Morbid Anatomy.—The mucosa is at first swollen and presents scattered tubercles, which seem to begin in the neighborhood of the blood-vessels. By their fusion small tuberculous masses arise, which caseate and finally ulcerate, leaving shallow irregular losses of substance. The ulcers are usually covered with a grayish exudation, and there is a general thickening of the mucosa about them, which is particularly marked upon the arytenoids. The ulcers may erode the true cords and finally destroy them, and passing deeply may cause perichondritis with necrosis and occasionally exfoliation of the cartilages. The disease may extend laterally and involve the pharynx, and downward over the mucous membrane, covering the cricoid cartilage toward the œsophagus. Above, it may reach the posterior wall of the pharynx, and in rare cases extend to the fauces and tonsils. The epiglottis may be entirely destroyed. There are rare instances in which cicatricial changes go on to such a degree that stenosis of the larynx is induced.

Symptoms.—The first indication is slight huskiness of the voice, which finally deepens to hoarseness, and in advanced stages there may be complete loss of voice. There is something very suggestive in the early hoarseness of tuberculous laryngitis. The attention may be directed to the lungs simply by the quality of the voice.

The cough is in part due to involvement of the larynx. Early in the disease it is not very troublesome, but when the ulceration is extensive it becomes husky and ineffectual. Of the symptoms, none is more aggravating than the dysphagia, which is met with particularly when the epiglottis is involved, and when the ulceration has extended to the pharynx. There is no

more distressing or painful complication in phthisis. In instances in which the epiglottis is in great part destroyed with each attempt to take food there are distressing paroxysms of cough, and even of suffocation.

With the laryngoscope there is seen early in the disease a pallor of the mucous membrane, which also looks thickened and infiltrated, particularly that covering the arytenoid cartilages. The ulcers are very characteristic. They are broad and shallow, with gray bases and ill-defined outlines. The vocal cords are infiltrated and thickened, and ulceration is very common.

The diagnosis is rarely difficult, as it is usually associated with well-marked pulmonary disease. In case of doubt the secretion from the base of an ulcer should be examined for bacilli.

Treatment.—The voice should not be used. In the early stages no method of treatment is more effectual. The ulcers should be sprayed and kept thoroughly cleansed with a solution of tannic acid, nitrate of silver, or sulphate of zinc. The insufflation, three times a day, of a powder of iodoform with morphia, after cleansing the ulcers with a spray, relieves the pain in a majority of the cases. Cocaine (4-per-cent. solution) applied with the atomizer will often enable the patient to swallow his food comfortably. There are, however, distressing cases of extensive laryngeal and pharyngeal ulceration in which even cocaine loses its good effects. When the epiglottis is lost the difficulty in swallowing becomes very great. Wolfenden states that this may be obviated if the patient hangs his head over the side of the bed and sucks milk through a rubber tube from a mug placed on the floor.

VI. SYPHILITIC LARYNGITIS

Syphilis attacks the larynx with great frequency. It may result from the inherited disease or as a secondary or tertiary manifestation of the acquired form.

Symptoms.—In secondary syphilis there is occasionally erythema of the larynx, which may go on to definite catarrh, but has nothing characteristic. The process may proceed to the formation of superficial whitish ulcers, usually symmetrically placed on the cords or ventricular bands. Mucous patches and condylomata are rarely seen. The symptoms are practically those of slight loss of voice with laryngeal irritation, as in the simple catarrhal form.

The tertiary laryngeal lesions are numerous and very serious. True gummata, varying in size from the head of a pin to a small nut, arise in the submucous tissue, most commonly at the base of the epiglottis. They go through the changes characteristic of these structures and may either break down, producing extensive and deep ulceration, or—and this is more characteristic of syphilitic laryngitis—in their healing form a fibrous tissue which shrinks and produces stenosis. The ulceration is apt to extend deeply and involve the cartilage, inducing necrosis and exfoliation, and even hæmorrhage from erosion of the arteries. Œdema may suddenly prove fatal. The cicatrices which follow the sclerosis of the gummata or the healing of the ulcers produce great deformity. The epiglottis, for instance, may be tied down to the pharyngeal wall or to the epiglottic folds, or even to the tongue; and eventually a stenosis results, which may necessitate tracheotomy.

The laryngeal symptoms of ~~inherited~~ syphilis have the usual course of these lesions and appear either early, within the first five or six months, or after puberty; most commonly in the former period. The gummatous infiltration leads to ulceration, most commonly of the epiglottis and in the ventricles, and the process may extend deeply and involve the cartilage. Cicatricial contraction may also occur.

The diagnosis of syphilis of the larynx is rarely difficult, since it occurs most commonly in connection with other symptoms of the disease.

Treatment.—The administration of constitutional remedies is the most important, and under mercury and iodide of potassium the local symptoms may rapidly be relieved. The tertiary laryngeal manifestations are always serious and difficult to treat. The deep ulceration is specially hard to combat, and the cicatrization may necessitate tracheotomy, or gradual dilatation, ~~as practiced by Schroetter.~~

C. DISEASES OF THE BRONCHI

I. ACUTE BRONCHITIS

Acute catarrhal inflammation of the bronchial mucous membrane is a very common disease, rarely serious in healthy adults, but very fatal in the old and in the young, owing to associated pulmonary complications. It is bilateral and affects either the larger and medium sized tubes or the smaller bronchi, in which case it is known as capillary bronchitis.

We shall speak only of the former, as the latter is part and parcel of broncho-pneumonia.

Etiology.—Acute bronchitis is a common sequel of "catching cold," and is often nothing more than the extension downward of an ~~ordinary coryza~~. It occurs most frequently in the changeable weather of early spring and late autumn. ~~Its association with cold is well indicated by the popular expression "cold on the chest."~~ It may prevail as an epidemic apart from influenza, of which it is an important feature.

Acute bronchitis is associated with many other affections, notably measles. It is by no means rare at the onset of typhoid fever and malaria. It is present also in asthma and whooping-cough. The subjects of spinal curvature are specially liable to the disease. The bronchitis of ~~Bright's~~ disease, gout, and heart-disease is usually a chronic form. It attacks persons of all ages, but most frequently the young and the old. There are individuals who have a special disposition to bronchial catarrh, and the slightest exposure is apt to bring on an attack. Persons who live an out-of-door life are usually less subject to the disease than those who follow sedentary occupations.

Bacteriology.—The pneumococcus is responsible for many cases both in young and old. The infection may follow pneumonia, and bronchitis may recur winter after winter, with the sputum showing an almost pure culture of the pneumococcus. ~~In one patient~~ These germs ~~have~~ persisted in the sputum for ~~seven~~ years, with an almost daily cough, aggravated in the winter. The influenza bacillus is ~~very~~ common and may be found alone or with streptococci. The *Micrococcus catarrhalis* is present in ~~a majority~~ of the ordinary

nasal affection /

Sol

Lubeck. pneumococcus v virulence in the great majority of cases.

By [illegible] fresh blood agar plates the organism can be determined in 95% of acute non-tuberculous infections of the respiratory tract in adults.

some.

cases, very often in combination with other organisms. Less frequently the staphylococci, colon bacillus, and typhoid bacilli have been found.

Morbid Anatomy.—The mucous membrane of the trachea and bronchi is reddened, congested, and covered with mucus and muco-pus, which may be seen oozing from the smaller bronchi, some of which are dilated. The finer changes in the mucosa consist in desquamation of the ciliated epithelium, swelling and œdema of the submucosa, and infiltration of the tissue with leucocytes. The mucous glands are much swollen.

Symptoms.—General.—The symptoms of an ordinary "cold" accompany the onset of an acute bronchitis. The coryza extends to the tubes, and may also affect the larynx, producing hoarseness, which in many cases is marked. A chill is rare, but there is invariably a sense of oppression, with heaviness and languor and pains in the bones and back. In mild cases there is scarcely any fever, but in severer forms the range is from 101° to 103° F. The bronchial symptoms set in with a feeling of tightness and rawness beneath the sternum and a sensation of oppression in the chest. The cough is rough at first, and often of a ringing character. It comes on in paroxysms which rack and distress the patient extremely. During the severe spells the pain may be very intense beneath the sternum and along the attachments of the diaphragm. At first the cough is dry and the expectoration scanty and viscid, but in a few days the secretion becomes muco-purulent and abundant, and finally purulent. With the loosening of the cough great relief is experienced. The sputum is made up largely of pus-cells, with a variable number of the large round alveolar cells, many of which contain carbon grains, while others have undergone the myelin degeneration.

Physical Signs.—The respiratory movements are not greatly increased in frequency unless the fever is high. There are instances, however, in which the breathing is rapid and when the smaller tubes are involved there is dyspnœa. On palpation the bronchial fremitus may often be felt. On auscultation in the early stage, piping sibilant râles are everywhere to be heard. They are very changeable, and appear and disappear with coughing. With the relaxation of the bronchial membranes and the greater abundance of the secretion, the râles change and become mucous and bubbling in quality. The bases of the lungs should be ~~carefully~~ examined each day, particularly in children and the aged.

Course.—The course of the disease depends on the conditions under which it arises. In healthy adults, by the end of a week the fever subsides and the cough loosens. In another week or ten days convalescence is fully established. In young children the chief risk is in the extension of the process downward. In measles and whooping-cough the ordinary bronchial catarrh is very apt to descend to the finer tubes, which become dilated and plugged with muco-pus, inducing areas of collapse, and finally broncho-pneumonia. This extension is indicated by changes in the physical signs. Usually at the base the râles are subcrepitant and numerous and there may be areas of defective resonance and of feeble or distant tubular breathing. In the aged and debilitated there are similar dangers if the process extends from the larger to the smaller tubes. In old age the bronchial mucosa is less capable of expelling the mucus, which is more apt to sag to the dependent parts and induce dilatation of the tubes with extension of the inflammation to the contiguous air-cells.

Diagnosis.—The diagnosis of acute bronchitis is rarely difficult. Although the mode of onset may be brusque and perhaps simulate pneumonia, yet the absence of dulness and blowing breathing, and the general character of the bronchial inflammation, render the diagnosis easy. The complication of broncho-pneumonia is indicated by the greater severity of the symptoms, particularly the dyspnœa, the changed color, and the physical signs.

Treatment.—In mild cases household measures suffice. The hot foot-bath, or the warm bath, a drink of hot lemonade, and a mustard plaster on the chest will often give relief. In severe cases the patient should be in bed; liquids should be taken freely. For the dry, racking cough, the symptom most complained of by the patient, Dover's powder is the best remedy. It is a popular belief that quinine, in full doses, will check an oncoming cold on the chest, but this is doubtful. It is a common custom when persons feel the approach of a cold to take a Turkish bath, and though the tightness and oppression may be relieved by it, there is in a majority of the cases great risk. Some of the severest cases of bronchitis which I have seen have followed this initial Turkish bath. No doubt, if the person could go to bed directly from the bath, its action would be beneficial, but there is great risk of catching "cold" in going home from the bath. Hydrotherapy is most useful in the form of compresses to the thorax or a wet pack. Relief is obtained from the unpleasant sense of rawness by keeping the air of the room saturated with moisture, and in this dry stage the old-fashioned mixture of the wines of antimony and ipecacuanha with liquor ammonii acetatis and nitrous ether is useful. If the pulse is very rapid, tincture of aconite may be given, particularly in the case of children. The use of inhalations, such as the compound tincture of benzoin, often gives relief. For the cough, when dry and irritating, opium should be freely used in the form of Dover's powder or paregoric. Of course, in the very young and the aged care must be exercised in the use of opium, particularly if the secretions are free; but for the distressing, irritative cough, which keeps the patient awake, opium in some form gives the only relief. Heroin is often helpful for this. As the cough loosens and the expectoration is more abundant, the patient becomes more comfortable. In this stage it is customary to ply him with expectorants of various sorts. Though useful occasionally, they should not be given as a matter of routine. A mixture of squill, ammonia, and senega is a favorite one with many practitioners at this stage. *Vaccine treatment* is not very successful, even when a single organism has been recovered.

In the acute bronchitis of children, if the amount of secretion is large and difficult to expectorate, or if there is dyspnœa and the color begins to get dusky, an emetic (a tablespoonful of ipecac wine) should be given at once and repeated if necessary.

II. CHRONIC BRONCHITIS

Etiology.—This affection may follow repeated attacks of acute bronchitis, but it is most commonly met with in chronic lung affections, heart-disease, aneurism of the aorta, gout, and renal disease. It is frequent in the aged: the young rarely are affected. Climate and season have an important influ-

Musical [rules]

ence. It is the cause of the winter cough of the aged, which recurs with regularity as the weather gets cold and changeable. Owing to the more uniform heating of the houses, it is much less common in Canada and in the United States than in England.

Morbid Anatomy.—The bronchial mucosa presents a great variety of changes, depending somewhat upon the disease with which chronic bronchitis is associated. In some cases the mucous membrane is very thin, so that the longitudinal bands of elastic tissue stand out prominently. The tubes are dilated, the muscular and glandular tissues are atrophied, and the epithelium is in great part shed.

In other instances the mucosa is thickened, granular, and infiltrated. There may be ulceration, particularly of the mucous follicles. Bronchial dilatations are not uncommon and emphysema is a constant accompaniment.

Symptoms.—In the form met with in old men, associated with emphysema, gout, or heart-disease, the chief symptoms are as follows: Shortness of breath, which may not be noticeable except on exertion. The patients "puff and blow" on going up hill or up a flight of stairs. This is due not so much to the chronic bronchitis itself as to associated emphysema or even to cardiac weakness. They complain of no pain. The cough is variable, changing with the weather and with the season. During the summer they may remain free, but each succeeding winter the cough comes on with severity and persists. There may be only a spell in the morning, or the chief distress is at night. The sputum in chronic bronchitis is very variable. In cases of the so-called dry catarrh there is no expectoration. Usually, however, it is abundant, mucopurulent, or distinctly purulent in character. There are instances in which the patient coughs up for years a thin fluid sputum. There is rarely fever. The general health may be good and the disease may present no serious features apart from the liability to induce emphysema and bronchiectasis. In many cases it is an incurable affection. Patients improve and the cough disappears in the summer time only to return during the winter months.

Physical Signs.—The chest is usually distended, the movements are limited, and the condition is often that which we see in emphysema. The percussion note is clear or hyperresonant. On auscultation, expiration is prolonged and wheezy and rhonchi of various sorts are heard—some high-pitched and piping, others deep-toned and snoring. Crepitant râles are common at the bases.

Clinical Varieties.—The description just given is of the ordinary chronic bronchitis which occurs in connection with emphysema and heart-disease and in many elderly men. There are certain forms which merit special description: (*a*) There is a form of chronic bronchitis in women, which comes on between the ages of twenty and thirty and may continue indefinitely without serious impairment of the health. In several cases the cough followed influenza, and there may be slight bronchiectasis.

(*b*) Bronchorrhœa.—Excessive bronchial secretion is met with under several conditions. It must not be mistaken for the profuse expectoration of bronchiectasis. The secretion may be very liquid and watery—*bronchorrhœa serosa*—and in extraordinary amount. More commonly, it is purulent though thin, and with greenish or yellow-green masses. It may be thick and uniform. This profuse bronchial secretion is usually a manifestation of chronic bron-

chitis, and may lead to dilatation of the tubes and ultimately to fetid bronchitis. In the young the condition may persist for years without impairment of health and without apparently damaging the lungs.

(*c*) Putrid Bronchitis.—Fetid expectoration is met with in connection with bronchiectasis, gangrene, abscess, or with decomposition of secretions within phthisical cavities and in an empyema which has perforated the lung. There are instances in which, apart from any of these states, the expectoration has a fetid character. The sputa are abundant, usually thin, grayish-white in color, and they separate into an upper fluid layer capped with frothy mucus and a thick sediment in which may sometimes be found dirty yellow masses the size of peas or beans—the so-called *Dittrich's* plugs. The affection is very rare apart from the above-mentioned conditions. In severe cases it leads to changes in the bronchial walls, pneumonia, and often to abscess or gangrene. Metastatic brain abscess has followed putrid bronchitis in a certain number of cases.

(*d*) Dry Catarrh.—The *catarrhe sec* of Laennec, a not uncommon form, is characterized by paroxysms of coughing of great intensity, with little or no expectoration. It is usually met with in elderly persons with emphysema, and is one of the most obstinate of all varieties of bronchitis.

Treatment.—Removal to a southern latitude may prevent the onset. In England the milder climate of Falmouth, Torquay, and Bournemouth is suitable for those who cannot go elsewhere. Egypt, southern France, southern California, and Florida furnish winter climates in which the subjects of chronic bronchitis live with the greatest comfort. With care chronic bronchitis may prove to be the slight ailment that, as Oliver Wendell Holmes says, promotes longevity.

The first endeavor is to ascertain, if possible, whether there are constitutional or local affections with which it is associated. In many instances the urine is found to be highly acid, perhaps slightly albuminous, and the arteries are stiff. In the form associated with this condition, sometimes called gouty bronchitis, the attacks seem related to the defective renal elimination, and to this condition the treatment should be first directed. In other instances there are heart-disease and emphysema. In the form occurring in old men much may be done in the way of prophylaxis. There is no doubt that with prudence even in the most changeable winter weather much may be done to prevent the onset of chronic bronchitis. Woollen undergarments should be used and especial care should be taken in the spring months not to change them for lighter ones before the warm weather is established.

Cure is seldom effected by medicinal remedies. There are instances in which iodide of potassium acts with remarkable benefit, and it should always be given a trial in cases of paroxysmal bronchitis of obscure origin. For the morning cough, bicarbonate of sodium (gr. xv, 1 gm.), chloride of sodium (gr. v, 0.3 gm.), spirits of chloroform (♏ v, 0.3 c. c) in anise water and taken with an equal amount of warm water will be found useful (Fowler). When there is much sense of tightness and fullness of the chest, the portable Turkish bath may be tried. When the secretion is excessive muriate of ammonia and senega are useful. Stimulating expectorants are contraindicated. When the heart is feeble, the combination of digitalis and strychnia is very beneficial. Turpentine, the old-fashioned remedy so warmly recommended by

the
is most important,

the Dublin physicians, has in many quarters fallen undeservedly into disuse. Preparations of tar, creosote, and terebene are sometimes useful. Of other balsamic remedies, sandal-wood, the compound tincture of benzoin, copaiba, balsam of Peru or tolu may be used. Inhalations of eucalyptus and of the spray of ipecacuanha wine are often very useful. If fetor be present, carbolic acid in the form of spray (1 to 2 per cent. solution) will lessen the odor, or thymol (1 to 1,000), but the intratracheal medication is the most efficient. After the larynx is anæsthetized with a 4 per cent. cocaine solution, inject with a suitable syringe about two drachms (8 c.c.) of olive oil, with gr. ½ (0.032 gm.) of iodoform, and gr. ⅛ (0.008 gm.) of morphia if there is irritating cough. For urgent dyspnœa with cyanosis, bleeding from the arm gives most relief.

III. BRONCHIECTASIS

Etiology.—The following excellent classification is given by Barty King:

- I. Bronchiolectasis
  - Acute
  - Chronic
- II. Bronchiectasis
  - A. Pure
    1. Chronic bronchitis
    2. Broncho-pneumonic
    3. Chronic pneumonic
    4. Pneumonic
    5. Pleuritic
  - B. Tuberculous
  - C. Traumatic
    1. Aneurism
    2. Tumor
    3. Foreign body
    4. Syphilis

In addition there is a congenital defect which Grawitz has described as *bronchiectasis universalis.*

Unquestionably the weakening of the bronchial wall is the most important, probably the essential, factor in inducing bronchiectasis, since the wall is then not able to resist the pressure of air in severe spells of coughing and in straining. In some instances the mere weight of the accumulated secretion may be sufficient to distend the terminal tubules, as is seen in compression of a bronchus by aneurism. Barty King lays great stress on pleural adherency as a factor in the initial dilatation of the tubes. The disease seems to have increased in frequency since the influenza epidemics of the past fifteen years. Of six consecutive cases in my wards in the session of 1904-05 from every one Boggs isolated the influenza bacillus.

Morbid Anatomy.—Two chief forms of bronchiectasis are recognized—the *cylindrical* and the *saccular*—which may exist together in the same lung. The condition may be general or partial. Universal bronchiectasis is always unilateral. It occurs in rare congenital cases and is occasionally seen as a sequence of interstitial pneumonia. The entire bronchial tree is represented by a series of sacculi opening one into the other. The walls are smooth and

possibly without ulceration or erosion except in the dependent parts. The lining membrane of the sacculi is usually smooth and glistening. The dilatations may form large cysts immediately beneath the pleura. Intervening between the sacculi is a dense cirrhotic lung tissue. The partial dilatations—the saccular and cylindrical—are common in chronic phthisis, particularly at the apex, in chronic pleurisy at the base, and in emphysema. Here the dilatation is more commonly cylindrical, sometimes fusiform. The bronchial mucous membrane is much involved and sometimes there is a narrowing of the lumen. Occasionally one meets with a single saccular bronchiectasis in connection with chronic bronchitis or emphysema. Some of these look like simple cysts, with smooth walls, without fluid contents. Bronchiolectasis as an acute condition may follow the infectious diseases, as in the cases described by Sharkey, Carr, and others. The chronic variety is a sequel of bronchitis in old subjects.

Histologically the bronchi which are the seat of dilatation show important changes. In the large, smooth dilatations the cylindrical is replaced by a pavement epithelium. The muscular layer is stretched, atrophied, and the fibres separated; the elastic tissue is also much stretched and separated. In the large saccular bronchiectases and in some of the cylindrical forms, due to retained secretions, the lining membrane is ulcerated. The contents of some of the larger bronchiectatic cavities are horribly fetid.

Symptoms.—There are *acute* cases, usually the bronchiolectasis of children; but a case in my wards of the broncho-pneumonic form died in six weeks from the onset. The bronchi of the lower lobes were dilated; there were areas of broncho-pneumonia and one or two spots of gangrene. The patient became hemiplegic, probably from abscess of the brain. In the limited dilatations of phthisis, emphysema, and chronic bronchitis the symptoms are in great part those of the original disease, and the condition often is not suspected during life.

In extensive saccular bronchiectasis the characters of the cough and expectoration are distinctive. The patient will pass the greater part of the day without any cough and then in a severe paroxysm will bring up a large quantity of sputum. Ten of my cases showed this symptom. Of 23 of my cases the amount for twenty-four hours was in 2 less than 100 c. c., in 11 from 100-300 c. c., in 2 almost 500 c. c., in 7 over 600 c. c. In one case with over one litre per day the cavities found were very small. Sometimes change of position will bring on a violent attack, probably due to the fact that some of the secretion flows from the dilatation to a normal tube. The daily spell of coughing is usually in the morning. The expectoration is in many instances very characteristic; it is grayish or grayish brown in color, fluid, purulent, with a peculiar acid, sometimes fetid, odor. Placed in a conical glass, it separates into a thick granular layer below and a thin mucoid intervening layer above, which is capped by a brownish froth. Microscopically it consists of pus-corpuscles, often large crystals of fatty acids, which are sometimes in enormous numbers over the field and arranged in bunches. Hæmatoidin crystals are sometimes present. Elastic fibres are seldom found except when there is ulceration of the bronchial walls. Tubercle bacilli are not present. In some cases, as in 10 of my series, the expectoration is very fetid and has all the characters of that described under fetid bronchitis. Nummular expectora-

cut down fluids = to 600 cc. = in/out day drink first freely. Then restrict again to 400. 300. or 200 for 3 days.

Some cases of asthma also helped.

Done

tion, such as comes from phthisical cavities, is not common. Hæmorrhage occurred in 14 out of 35 cases analyzed by Fowler, in 17 of my 24 cases, slight in 8, and extreme in 3. Arthritis may occur, and it is one of the conditions with which the pulmonary osteo-arthropathy is commonly associated. There is a remarkable association of bronchiectasis with abscess of the brain. Among 13,700 autopsies at the London Hospital and the Brompton Hospital there were 19 instances of cerebral abscess with pulmonary disease, usually bronchiectasis (Schörstein).

Diagnosis.—In the extensive sacculated forms, unilateral and associated with interstitial pneumonia or chronic pleurisy, the diagnosis is easy. There is contraction of the side, which in some instances is not at all extreme. The cavernous signs may be chiefly at the base and may vary according to the condition of the cavity, whether full or empty. There may be the most exquisite amphoric phenomena and loud resonant râles. The condition persists for years and is not inconsistent with a tolerably active life. The patients frequently show signs of marked embarrassment of the pulmonary circulation. There is a cyanosis on exertion, the finger-tips are clubbed, and the nails incurved. A condition very difficult to distinguish from bronchiectasis is a limited pleural cavity communicating with a bronchus. The X-ray examination is an important aid in diagnosis.

Treatment.—Medical treatment is not satisfactory, since it is impossible to heal the cavities. Postural treatment is important, and the most favorable position should be studied for each patient. Sleeping with the head low favors "drainage." I have practiced the injection of antiseptic fluids in some instances with benefit. Intratracheal injections have been recommended of late. With a suitable syringe a drachm may be injected twice a day of the following solution: Menthol 10 parts, guaiacol 2 parts, olive oil 88 parts. Or better still when the odor is very offensive iodoform in olive oil. The creosote vapor bath may be given in a small room. The patient's eyes must be protected with well-fitting goggles, and the nostrils stuffed with cotton-wool. Twenty to thirty drops of creosote are poured upon water in a saucer and vaporized by placing the saucer over a spirit lamp. At first the vapor is very irritating and disagreeable, but the patient gets used to it. The bath should be taken at first every other day for fifteen minutes, then gradually increased to an hour daily. The treatment should be continued for three months. I can recommend it as a most satisfactory method. In suitable cases, as when there is a single large cavity, drainage of the cavities may be attempted, particularly if the patient is in fairly good condition. Resection of the overlying ribs has been employed. For the fetid secretion turpentine may be given, or terebene, and inhalations of carbolic acid or thymol used.

IV. BRONCHIAL ASTHMA

Asthma is a term which has been applied to various conditions associated with dyspnœa—hence the names cardiac and renal asthma—but its use should be limited to the affection known as bronchial or spasmodic asthma.

Etiology.—All writers agree that there is in a majority of cases of bronchial asthma a strong neurotic element. Many regard it as a neurosis in

which, according to one view, spasm of the bronchial muscles, according to the other turgescence of the mucosa, results from disturbed innervation, pneumogastric or vaso-motor. Of the numerous theories the following are the most important:

(1) That it is due to spasm of the bronchial muscles, a theory which has perhaps the largest number of adherents. The original experiments of C. J. B. Williams, upon which it is largely based, have been confirmed by Brodie.

(2) That the attack is due to swelling of the bronchial mucous membrane —fluctionary hyperæmia (Traube), vaso-motor turgescence (Weber), diffuse hyperæmic swelling (Clark).

(3) That in many cases it is a special form of inflammation of the smaller bronchioles—*bronchiolitis exudativa* (Curschmann). Other theories which may be mentioned are that the attack depends on spasm of the diaphragm or on reflex spasm of all the inspiratory muscles.

As already mentioned, the so-called hay fever is an affection which has many resemblances to bronchial asthma, with which the attacks may alternate. In the suddenness of onset and in many of their features these diseases have a great similarity and differ only in site, as suggested by Sir Andrew Clark and generally acknowledged by specialists. Making due allowance for anatomical differences, if the structural changes occurring in the nasal mucous membrane during an attack of hay fever were to occur also in various parts of the bronchial mucosa, their presence there would afford a complete and adequate explanation of the facts observed during a paroxysm of bronchial asthma (Clark). With this statement I fully agree, but the observations of Curschmann have directed attention to a feature in asthma which has been neglected; namely, that in a majority of the cases it is associated with an exudation, such as might be supposed to come from a turgescent mucosa and which is of a very characteristic and peculiar character. The hyperæmia and swelling of the mucosa and the extremely viscid, tenacious mucus explain well the hindrance to inspiration and expiration and also the quality of the râles. An œdema of the angio-neurotic type has been described in the hands and arms in asthma.

Some general facts with reference to etiology may be mentioned. The affection sometimes runs in families, particularly those with irritable and unstable nervous systems. The attack may be associated with neuralgia or, as Salter mentions, even alternate with epilepsy. Men are more frequently affected than women. The disease often begins in childhood and sometimes lasts until old age. For years asthmatic attacks may follow whooping-cough. One of its most striking peculiarities is the *bizarre* and extraordinary variety of circumstances which at times induce a paroxysm. Among these local conditions climate or atmosphere is most important. A person may be free in the city and invariably suffer from an attack when he goes into the country, or into one special part of the country. Such cases are by no means uncommon. Breathing the air of a particular room or a dusty atmosphere may bring on an attack. Odors, particularly of flowers and of hay, or emanations from animals, as the horse, dog, or cat, may at once cause an outbreak. Fright or violent emotion of any sort may bring on a paroxysm. Uterine and ovarian troubles were formerly thought to induce attacks and may do so in rare instances. Diet, too, has an important influence, and in persons subject to the

Some

Inspiratory stream of diaphragm

Protein sensitization (anaphylaxis)

or on protein sensitization.

Walker J. Med. Res. Jan. 17.

Sensitization to bacterial proteins - abscess action probably to organisms forms in resp. tract.

—

Nicotine sensitizes on anaphylactic reaction to egg white tested by skin reaction.

anaphylaktic reaction in lung?

sometimes an anaph. to egg albumin with shortness

of breath & wheezing.

carbon. here comes by horse serum.

disease severe paroxysms may be induced by overloading the stomach, or by taking certain articles of food. Chronic cases, in which the attacks recur year after year, gradually become associated with emphysema, and every fresh "cold" induces a paroxysm. And, lastly, many cases of bronchial asthma are associated with affections of the nose, particularly with hypertrophic rhinitis and nasal polypi.

Briefly stated, then, bronchial asthma is a neurotic affection, characterized by hyperæmia and turgescence of the mucosa of the smaller bronchial tubes and a peculiar exudate of mucin. The attacks may be due to direct irritation of the bronchial mucosa or may be induced, reflexly, by irritation of the nasal mucosa, and indirectly, too, by reflex influences, from stomach, intestines, or genital organs. It is important to remember that in the subjects of asthma to whom injections of diphtheria or other antitoxins are given anaphylaxis may be induced with a rapidly fatal termination.

Symptoms.—Premonitory sensations precede some attacks, such as chilly feelings, a sense of tightness in the chest, flatulence, the passage of a large quantity of urine, or great depression of spirits. Nocturnal attacks are common. After a few hours' sleep, the patient is aroused with a distressing sense of want of breath and a feeling of great oppression in the chest. Soon the respiratory efforts become violent, all the accessory muscles are brought into play, and in a few minutes the patient is in a paroxysm of the most intense dyspnœa. The face is pale, the expression anxious, speech is impossible, and in spite of the most strenuous inspiratory efforts very little air enters the lungs. Expiration is prolonged and also wheezy. The number of respirations, however, is not much increased. The asthmatic fit may last from a few minutes to several hours. When severe, the signs of defective aëration soon appear, the face becomes bedewed with sweat, the pulse is small and quick, the extremities get cold, and just as the patient seems to be at his worst the breathing begins to get easier, and often with a paroxysm of coughing relief is obtained and he sinks exhausted to sleep. The relief may be but temporary and a second attack may soon come on. In a majority of the cases even in the intervals between the asthmatic fits the respiration is somewhat embarrassed. The cough is at first very tight and dry and the expectoration is tenacious. Emphysema of the neck may occur during the violent coughing spells. Urticaria may break out over the whole body during an attack, or, as in one patient, may be confined to the skin of the interscapular regions.

The PHYSICAL SIGNS during an attack are very characteristic. On inspection the thorax looks enlarged, barrel-shaped, and is fixed, the amount of expansion being altogether disproportionate to the intensity of the inspiratory movements. The diaphragm is lowered and moves but slightly. Inspiration is short and quick, expiration prolonged. Percussion may not reveal any special difference, but there is sometimes marked hyperresonance, particularly in patients who have had repeated attacks.

On auscultation, with inspiration and expiration, there are innumerable sibilant and sonorous râles of all varieties, piping and high-pitched, low-pitched and grave. Later in the attack there are moist râles.

The *sputum* is quite distinctive, unlike that which occurs in any other affection. Early in the attack it is brought up with great difficulty and is in the form of rounded gelatinous masses, the so-called *"perles"* of Laennec.

Though ball-like, they can be unfolded and really represent moulds in mucus of the smaller tubes. The entire expectoration may be made up of these somewhat translucent-looking pellets, floating in a small quantity of thin mucus. Some of them are opaque. Often with the naked eye a twisted spiral character can be seen, particularly if the sputum is spread on a glass with a black background. Microscopically, many of these pellets have a spiral structure, which renders them among the most remarkable bodies met with in sputum. It is not a little curious that they should have been practically overlooked until described by Curschmann. Under the microscope the spirals are of two forms. In one there is simply a twisted, spirally arranged filament of mucin, in which are entangled leucocytes, the majority of which are eosinophiles. The twist may be loose or tight. The second form is much more peculiar. In the centre of a tightly coiled skein of mucin fibrils with a few scattered cells is a filament of extraordinary clearness and translucency, probably composed of transformed mucin. These spirals are doubtless formed in the finer bronchioles and constitute the product of an acute bronchiolitis. It is difficult to explain their spiral nature. I do not know of any observations upon the course of the currents produced by the ciliated epithelium in the bronchi, but it is quite possible that their action may be rotatory, in which case, particularly when combined with spasm of the bronchial muscles, it is possible to conceive that the mucus formed in the tube might be compelled to assume a spiral form. Within two or three days the sputum changes entirely in character; it becomes muco-purulent and Curschmann's spirals are no longer to be found. They occur in all instances of true bronchial asthma in the early period of the attack. I have never seen the true spirals either in bronchitis or pneumonia. There are, in addition, in many cases, the pointed, octahedral crystals described by Leyden and sometimes called asthma crystals. They are identical with the crystals found in the semen and in the blood in leukæmia. At one time they were supposed, by their irritating character, to induce the paroxysms. Eosinophiles in the blood are enormously increased in asthma—to 25 or 35 per cent. of the leucocytes, or even to 53.6 per cent. in one case.

Course.—This is very variable. In severe attacks the paroxysms recur for three or four nights or even more, and in the intervals and during the day there may be wheezing and cough. Early in the disease the patient may be free in the morning, without cough or much distress, and the attacks may appear at first to be of a purely nervous character. In the long-standing cases emphysema almost invariably develops and, while the pure asthmatic fits diminish in frequency the chronic bronchitis and shortness of breath become aggravated.

We have no knowledge of the morbid anatomy of true asthma. Death during the attack is unknown. In long-standing cases the lesions are those of chronic bronchitis and emphysema.

Treatment.—The asthmatic attack usually demands immediate and prompt treatment, and remedies should be administered which experience has shown are capable of relieving the condition of the bronchial mucosa. A few whiffs of chloroform will produce prompt though temporary relaxation. In a child with very severe attacks, resisting all the usual remedies, the treatment by chloroform gave immediate and finally permanent relief. Hypodermic injec-

Anschaf

adrenalin | exclude organic meat
Bovine before giving.

Recurrent rhinitis + sibilant bronchitis - with asthmatic attacks
due often found to inhalation -

Dietary attack = biscuits, butter, eggs + raw fruits to be avoided

allow - beef, mutton, fresh fish, cereals, bread, vegs (except Shamrock
rhubarb, tomatoes, salads) cooked fruits - skins ... ick tomatoes
often 2 months. add 1 egg a day - take carbon meals
take also raw fruits (but wrapped)

tuberculosis

tions of pilocarpin (gr. 1/8, 0.008 gm.) will sometimes relax the mucosa in the profuse sweating. Perles of nitrite of amyl may be broken on the handkerchief or from two to five drops of the solution may be placed upon cotton-wool and inhaled. Strong stimulants given hot or a dose of spirit of chloroform in hot whisky will sometimes induce relaxation. More permanent relief is given by the hypodermic injection of morphia or of morphia and strychnine combined. In obstinate and repeatedly recurring attacks this has proved a very satisfactory plan. The sedative antispasmodics, such as belladonna, henbane, stramonium, and lobelia, may be given in solution or used in the form of cigarettes. Nearly all the popular remedies either in this form or in pastilles contain some plant of the order *solanaceæ*, with nitrate or chlorate of potash. Excellent cigarettes are now manufactured and asthmatics try various sorts, since one form benefits one patient, another form another patient. Nitre paper made with a strong solution of nitrate of potash is very serviceable. Filling the room with the fumes of this paper prior to retiring will sometimes ward off a nocturnal attack. I have known several patients to whom tobacco smoke inhaled was quite as potent as the prepared cigarettes.

Cauterization of the mucous membrane of the nose has given great relief, particularly in cases with swelling and irritation. The use of compressed air in the pneumatic cabinet is very beneficial; oxygen inhalations may also be tried. In preventing the recurrence of the attacks there is no remedy so useful as iodide of potassium, which sometimes acts like a specific. From 10 to 20 grains (0.6 to 1.3 gm.) three times a day is usually sufficient. Persistent hydrotherapy is often of value.

Particular attention should be paid to the diet of asthmatic patients. A rule which experience generally compels them to make is to take the heavy meals in the early part of the day and not retire to bed before gastric digestion is completed. As the attacks are often induced by flatulency, the carbohydrates should be restricted. Coffee is a more suitable drink than tea. In respect to climate it is very difficult to lay down rules for asthmatics. The patients are often much better in the city than in the country. The high and dry altitudes are certainly more beneficial than the sea-shore; but in the protracted cases, with emphysema as a secondary complication, the rarefied air of high altitudes is not advantageous. In young persons I have known a residence for six months in Florida or southern California, to be followed by prolonged freedom from attacks. Egypt is a peculiarly satisfactory winter climate.

V. FIBRINOUS BRONCHITIS

(*Plastic or Croupous Bronchitis*)

Definition.—An acute or chronic affection, characterized by the formation in certain of the bronchial tubes of fibrinous casts, which are expelled in paroxysms of dyspnœa and cough.

In several diseases fibrinous moulds of the bronchi are formed, as in diphtheria (with extension into the trachea and bronchi), in pneumonia, and occasionally in phthisis—conditions which, however, have nothing to do with true fibrinous bronchitis. These casts are not to be confounded with the blood-casts which occur occasionally in hæmoptysis.

Clinical Description.—Bettman, in reporting a case which occurred in Whitridge Williams's obstetrical clinic at the Johns Hopkins Hospital, analyzed all the cases from the literature since 1869, grouping them into different classes. The first and most important is *chronic idiopathic fibrinous bronchitis*. It is a rare affection. Of 27 cases, 15 were in males. It is most common at the middle period of life. The attacks may occur at definite intervals for months or years. The form and size of the casts may be identical at each attack as though each time precisely the same bronchial area was involved. The expectoration of the casts is associated with paroxysms of dyspnœa and coughing, which occur at longer or shorter intervals. Fever and hæmoptysis may be present during the attack. Physical signs usually indicate the portion of the lung affected, as there are suppressed breath sounds and numerous râles on coughing. A very dry râle, called the "*bruit de drapeau*," has been described, caused by the vibration of a loosened portion of the cast.

In five cases there were skin lesions. Tuberculosis is sometimes present. The casts are usually rolled up and mixed with mucus and blood. When unrolled they are large white branching structures. The main stem may be as thick as the little finger. From the consistency and appearance they have been described as fibrinous, but they consist mainly of mucin. On cross-section they show a concentrically stratified structure, with leucocytes and alveolar epithelium. Leyden's crystals and ~~Von~~ Curschmann's spirals are sometimes found, and in Bettman's case there were protozoan-like bodies. Death occurred in only one case of the series.

There is a very remarkable *acute form*, of which Bettman collected 15 cases. It comes on most frequently during some fever, as typhoid, pneumonia, or the eruptive fevers. After a preliminary bronchitis the dyspnœa increases, and then the casts are coughed up. Chills and fever have been present. Four of the 15 cases proved fatal, and the casts were found *in situ*. It is much more serious than the chronic form. There may be casts expectorated which have not the arborescent structure of the true fibrinous moulds, but which come from a single tube or its bifurcation. Sometimes they are very small and "tail off" into true spirals.

Fibrinous casts are expectorated in connection with chronic heart-disease ~~(10 cases)~~ and in pulmonary tuberculosis ~~(14 cases)~~, in the latter disease usually late in the course and of unfavorable moment. In the albuminous expectoration following tapping of a pleural exudate fibrinous casts have been coughed up.

In hæmoptysis blood-casts may be expectorated, and they are not to be confounded with the casts of true fibrinous bronchitis which may be coughed up with profuse hæmorrhage.

In pneumonia small fibrinous plugs are not uncommon in the sputum, and in a few rare instances quite large moulds of the tubes may be coughed up.

The mycelium of *Aspergillus fumigatus* may form membranous casts in the bronchi. ~~I~~ reported an instance of the kind in which a small partial mould of this kind was expectorated, and there is on record a case in which for long periods membranes composed of this fungus were coughed up in attacks of dyspnœa.

Pathology.—~~The pathology of the disease~~ is obscure. The membrane is identical with that to which the term croupous is applied, and the obscurity

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relates not so much to the mechanism of the production, which is probably the same as in other mucous surfaces, as to the curious limitation of the affection to certain bronchial territories and in the chronic form to the remarkable recurrence at stated or irregular intervals throughout a period of many years.

In the fatal cases the bronchial mucous membrane may be found injected or pale. In Biermer's case the epithelial lining was intact beneath the cast, but in that of Kretschy the bronchi were denuded of their epithelium. Emphysema is almost invariably present. Evidences of recent or antecedent pleurisy are sometimes found. Model, in an article published from Bäumler's clinic, states that tuberculosis was present in 10 out of 21 autopsies.

Treatment.—In the acute cases the treatment should be that of ordinary acute bronchitis. We know of nothing which can prevent the recurrence of the attacks in the chronic form. In the uncomplicated cases there is rarely any danger during the paroxysm, even though the symptoms may be most distressing and the dyspnœa and cough very severe. Inhalations of ether, steam, or atomized lime-water aid in the separation of the membranes. Waldenberg employed the last remedy with success in one case. Ewart recommends intratracheal injections of olive oil. Pilocarpine might be useful, as in some instances it increases the bronchial secretion. The employment of emetics may be necessary, and in some cases they are effective in promoting the removal of the casts.

D. DISEASES OF THE LUNGS

I. CIRCULATORY DISTURBANCES IN THE LUNGS

Congestion.—There are two forms of congestion of the lungs—active and passive.

1. Active Congestion of the Lungs.—Much doubt and confusion still exist on this subject. French writers, following Woillez, regard it as an independent primary affection (*maladie de Woillez*), and in their dictionaries and text-books allot much space to it. English and American authors more correctly regard it as a symptomatic affection. Active fluxion to the lungs occurs with increased action of the heart, and when very hot air or irritating substances are inhaled. In diseases which interfere locally with the circulation the capillaries in the adjacent unaffected portions may be greatly distended. The importance, however, of this collateral fluxion, as it is called, is probably exaggerated. In a whole series of pulmonary affections there is this associated congestion—in pneumonia, bronchitis, pleurisy, and tuberculosis.

The symptoms of active congestion of the lungs are by no means definite. The description given by Woillez and by other French writers is of an affection which is difficult to recognize from anomalous or larval forms of pneumonia. The chief symptoms described are initial chill, pain in the side, dyspnœa, moderate cough, and temperature from 101° to 103° F. The physical signs are defective resonance, feeble breathing, sometimes bronchial in character, and fine râles. A majority of clinical physicians would undoubtedly class such cases under inflammation of the lung. In many epidemics the abnormal and larval forms are specially prevalent.

The occurrence of an intense and rapidly fatal congestion of the lung, following extreme heat or cold or sometimes violent exertion, is recognized by some authors. Renforth, the oarsman, is said to have died from this cause during a race at Halifax. Leuf has described cases in which, in association with drunkenness, exposure, and cold, death occurred suddenly, or within twenty-four hours, the only lesion found being an extreme, almost hæmorrhagic, congestion of the lungs. It is by no means certain that in these cases death really occurs from pulmonary congestion in the absence of specific statements with reference to the coronary arteries and the heart.

2. Passive Congestion.—Two forms of this may be recognized, the mechanical and the hypostatic.

(*a*) *Mechanical congestion* occurs whenever there is an obstacle to the return of the blood to the heart. It is a common event in many affections of the left heart. The lungs are voluminous, russet brown in color, cutting and tearing with great resistance. On section they show at first a brownish red tinge, and then the cut surface, exposed to the air, becomes rapidly of a vivid red color from oxidation of the abundant hæmoglobin. This is the condition known as *brown induration* of the lung. Histologically it is characterized by (*i*) great distention of the alveolar capillaries; (*ii*) increase in the connective-tissue elements of the lung; (*iii*) the presence in the alveolar walls of many cells containing altered blood-pigment; (*iv*) in the alveoli numerous epithelial cells containing blood-pigment in all stages of alteration, which are also found in great numbers in the sputum.

It occasionally happens that this mechanical hyperæmia of the lung results from pressure by tumors. So long as compensation is maintained the mechanical congestion of the lung in heart disease does not produce any symptoms, but with enfeebled heart action the engorgement becomes marked ~~and there are~~ dyspnœa, cough, and expectoration, ~~with~~ the characteristic alveolar cells.

(*b*) *Hypostatic Congestion.*—In fevers and adynamic states generally it is very common to find the bases of the lungs deeply congested, a condition induced partly by the effect of gravity, the patient lying recumbent in one posture for a long time, but chiefly by weakened heart action. That it is not an effect of gravity alone is shown by the fact that a healthy person may remain in bed an indefinite time without its occurrence. The posterior parts of the lung are dark in color and engorged with blood and serum; in some instances to such a degree that the alveoli no longer contain air and portions of the lung sink in water. The terms *splenization* and hypostatic pneumonia have been given to these advanced grades. It is a common affection in protracted cases of typhoid fever and in long debilitating illness. In ascites, meteorism, and abdominal tumors the bases of the lungs may be compressed and congested. In this connection must be mentioned the form of passive congestion met with in injury to, and organic disease of, the brain. In cerebral apoplexy the bases of the lungs are deeply engorged, not quite airless, but heavy, and on section drip with blood and serum. ~~I have twice seen~~ This condition, in an extreme grade throughout the lungs in death from morphia poisoning. In some instances the lung tissue has a blackish, gelatinous, infiltrated appearance, almost like diffuse pulmonary apoplexy. Occasionally this congestion is most marked in, and even confined to, the hemiplegic side. In prolonged coma the hypo-

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ap Increased production of tissue fluid — cardiac failure most important cause.
1) Increased intracapillary pressure
2) " permeability of capsule
3) Hydraemia.

Factors of removal — Lymphatic drainage.
1) Lymph stagnation.
2) Diminished capill. absorption.
3) Increased venous pressure

Tr. Cardiac failure — digitalis group. aspirin. Strophanthin. 1/200 - 1/100 mg iv

2) nitroglycerine — doubtful.
3) venesection
4) Resp. insufficiency — a) alkalosis — doubtful.
b) asphyxia — some use strych — ?
c) oxygen — useful in some.
d) artificial resp.
e) coramine — inhalation
f) lobeline head low
g) morphine
h) caffeine.

Morphine useful

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Turn over

static congestion may be associated with patches of consolidation, due to the aspiration of portions of food into the air-passages.

The symptoms of hypostatic congestion are not at all characteristic, and the condition has to be sought for by careful examination of the bases of the lungs, when slight dulness, feeble, sometimes blowing, breathing and liquid râles can be detected.

TREATMENT.—The treatment of congestion of the lungs is usually that of the condition with which it is associated. In the intense pulmonary engorgement, which may possibly occur primarily, and which is met with in heart disease and emphysema, free bleeding should be practiced. From 20 to 30 ounces of blood should be taken ~~from the arm, and if the blood does not flow freely and the condition of the patient is desperate aspiration of the right auricle may be performed~~.

Œdema.—In all forms of intense congestion of the lungs there is a transudation of serum from the engorged capillaries chiefly into the air-cells, but also into the alveolar walls. Not only is it very frequent in congestion, but also with inflammation, with new growths, infarcts, and tubercles. When limited to the neighborhood of an affected part, the name collateral œdema is sometimes applied to it.

Acute œdema is met with: (1) in the infections; (2) in Bright's disease; (3) in heart disease, particularly angina pectoris, myocarditis, and valve lesions; (4) in arterio-sclerosis; (5) pregnancy; (6) angio-neurotic œdema, and (7) as a complication of the epileptic fit. The theory most generally accepted is that of Welch, whose experiments seemed to indicate that pulmonary œdema is due to a disproportionate weakness of the left ventricle, so that the blood accumulates in the lung capillaries until transudation occurs. ~~Such weakness may be brought about by paralysis or by spasm of the left ventricle.~~ Others regard it as an effect of disturbance in the vasomotor mechanism of the lungs. In some cases there are recurring attacks of acute œdema.

Anatomically the lung is anæmic, heavy, sodden, pits on pressure, and on section a large quantity of clear or blood-tinged serum flows out. It may have in places a gelatinous aspect.

SYMPTOMS.—The onset is sudden with a feeling of oppression and pain in the chest and rapid breathing which soon becomes dyspnœic or orthopnœic. There may be an incessant short cough and a copious frothy, sometimes blood-tinged, expectoration, which may be expelled in a gush from the mouth and nose. The face is pale and covered with a cold sweat; the pulse is feeble and the heart's action weak. Over the entire chest may be heard piping and bubbling râles. The attack may be fatal in a few hours or it may persist for twelve or twenty-four hours and then pass off. Steven, of Glasgow, has reported a case with 72 attacks in two and a half years. I have seen this recurrent form in angina pectoris, each paroxysm of which was associated with intense dyspnœa and all the features of acute œdema of the lungs.

Bleeding should be practiced at once and is often most helpful. Dry cupping may be tried. ~~One of my~~ patients ~~had great~~ relief from inhalations of chloroform. Oxygen may be used. Atropine hypodermically (gr. 1/100, 0.00065 gm.) is sometimes of value. If there is much agitation and sense of impending death, morphia may be given hypodermically.

Pulmonary Hæmorrhage.—This occurs in two forms—*broncho-pulmonary hæmorrhage*, sometimes called bronchorrhagia, in which the blood is poured ~~out~~ into the bronchi and is expectorated, and *pulmonary apoplexy* or pneumorrhagia, in which the hæmorrhage takes place into the air-cells and the lung tissue.

1. Broncho-pulmonary Hæmorrhage; Hæmoptysis.—Spitting of blood, to which the term hæmoptysis should be restricted, results from a variety of conditions, among which the following are the most important: (*a*) In young healthy persons hæmoptysis may occur without warning, and after continuing for a few days disappear and leave no ill traces. There may be at the time of the attack no physical signs indicating pulmonary disease. In such cases good health may be preserved for years and no further trouble occur. These cases are not very uncommon, but in spite of the good health tuberculosis ~~may~~ be suspected. In Ware's important contribution, ~~to this subject~~, of 386 cases of hæmoptysis noted in private practice 62 recovered and pulmonary disease did not subsequently develop in them. (*b*) *Hæmoptysis in pulmonary tuberculosis*, which is considered on page 194. (*c*) In connection with certain diseases of the lung, as pneumonia (in the initial stage) and cancer, occasionally in gangrene, abscess, and bronchiectasis. (*d*) In many heart affections, particularly mitral lesions. It may be profuse and recur at intervals for years. (*e*) ***In ulcerative affections of the larynx, trachea, or bronchi.*** Sometimes the hæmorrhage is profuse and rapidly fatal, as when the ulcer erodes a large branch of the pulmonary artery, an accident which ~~I have known to~~ happen in a case of chronic bronchitis with emphysema. (*f*) *Aneurism* is an occasional cause of hæmoptysis. It may be sudden and rapidly fatal when the sac bursts into the air-passages. Slight bleeding may continue for weeks or months, due to pressure on the mucous membrane or erosion of the lung; or in some cases the sac "weeps" through the exposed laminæ of fibrin. (*g*) *Vicarious hæmorrhage*, which occurs in rare instances in cases of interrupted menstruation. The instances are well authenticated. Flint mentions a case which he had had under observation for four years, and Hippocrates refers to it in the aphorism, "Hæmoptysis in a woman is removed by an eruption of the menses." Periodical hæmoptysis has also been met with after the removal of both ovaries. Even fatal hæmorrhage has occurred from the lung during menstruation when no lesion was found to account for it. (*h*) There is a form of *recurring hæmoptysis in arthritic subjects* to which Sir Andrew Clark ~~has~~ called special attention and which ~~also~~ is described by French writers. ~~The cases~~ occur in persons over fifty years of age who usually present signs of the arthritic diathesis. It rarely leads to fatal issue and subsides without inducing pulmonary changes. (*i*) Hæmoptysis occurs sometimes *in malignant fevers* and *in purpura hæmorrhagica*. Lastly, there is endemic hæmoptysis, due to the bronchial fluke, an affection ~~which is~~ confined to parts of China and Japan.

Symptoms.—Hæmoptysis sets in, as a rule, suddenly. Often without warning the patient experiences a warm, saltish taste as the mouth fills with blood. Coughing is usually induced. There may be only an ounce or so brought up before the hæmorrhage stops, or the bleeding may continue for days, the patient bringing up small quantities. In other instances, particularly when a large vessel is eroded or an aneurism bursts, the amount is large, and the

patient, after a few attempts at coughing, shows signs of suffocation and death is produced by inundation of the bronchial system. Fatal hemorrhage even may occur into a large cavity in a patient debilitated by phthisis without the production of hæmoptysis. I dissected a case of this kind at the Philadelphia Hospital. The blood from the lungs generally has characters which render it readily distinguishable from the blood which is vomited. It is alkaline in reaction, frothy, and mixed with mucus, and when coagulation occurs air-bubbles are present in the clot. Blood-moulds of the smaller bronchi are sometimes seen. Patients can usually tell whether the blood has been brought up by coughing or by vomiting, and in a majority of cases the history gives important indications. In paroxysmal hæmoptysis connected with menstrual disturbances the practitioner should see that the blood is actually coughed up, since deception may be practiced. The spurious hæmoptysis of hysteria is considered with that disease. Naturally, the patient is at first alarmed at the occurrence of bleeding, but, unless very profuse, as when due to rupture of an aortic aneurism in a pulmonary cavity, the danger is rarely immediate. The attacks, however, are apt to recur for a few days and the sputum may remain blood-tinged for a longer period. In the great majority of cases the hæmorrhage ceases spontaneously. It should be remembered that some of the blood may be swallowed and produce vomiting, and, after a day or two, the stools may be dark in color. It is not advisable to examine the chest during an attack of hæmoptysis.

2. Pulmonary Apoplexy; Hæmorrhagic Infarct.—In this condition the blood is effused into the air-cells and interstitial tissue. It is usually diffuse, the parenchyma not being broken, as is the brain tissue in cerebral apoplexy. Sometimes, in disease of the brain, in septic conditions, and in the malignant forms of fevers, the lung tissue is uniformly infiltrated with blood and has, on section, a black, gelatinous appearance.

As a rule, the hæmorrhage is limited and results from the blocking of a branch of the pulmonary artery either by a thrombus or an embolus. The condition is most common in chronic heart-disease. Although the pulmonary arteries are terminal ones, blocking is not always followed by infarction; partly because the wide capillaries furnish sufficient anastomosis, and partly because the bronchial vessels may keep up the circulation. The infarctions are chiefly at the periphery of the lung, usually wedge-shaped, with the base of the wedge toward the surface. When recent, they are dark in color, hard and firm, and look on section like an ordinary blood-clot. Gradual changes go on, and the color becomes a reddish brown. The pleura over an infarct is usually inflamed. A microscopic section shows the air-cells to be distended with red blood corpuscles, which may also be in the alveolar walls. The infarcts are usually multiple and vary in size from a walnut to an orange. Very large ones may involve the greater part of a lobe. In the artery passing to the affected territory a thrombus or an embolus is found. The globular thrombi, formed in the right auricular appendix, play an important part in the production of hæmorrhagic infarction. In many cases the source of the embolus can not be discovered, and the infarct may have resulted from thrombosis in the pulmonary artery, but, as before mentioned, it is not infrequent to find total obstruction of a large branch of a pulmonary artery without hæmorrhage into the corresponding lung area. The further history of an

infarction is variable. It is possible that in some instances the circulation is re-established and the blood removed. More commonly, if the patient lives, the usual changes go on in the extravasated blood and ultimately a pigmented, puckered, fibroid patch results. Sloughing may occur with the formation of a cavity. Occasionally gangrene results. In a case at the University Hospital, Philadelphia, a gangrenous infarct ruptured and produced fatal pneumothorax.

The *symptoms* of pulmonary apoplexy are by no means definite. The condition may be suspected in chronic heart-disease when hæmoptysis occurs, particularly in mitral stenosis, but the bleeding may be due to the extreme engorgement. When the infarcts are very large, and particularly in the lower lobe, in which they most commonly occur, there may be signs of consolidation with blowing breathing and a pleuritic friction.

Treatment of Pulmonary Hæmorrhage.—The pressure within the pulmonary artery is considerably less than that in the aortic system. The system is under vaso-motor control, but our knowledge of the mutual relations of pressure in the aorta and in the pulmonary artery, under varying conditions, is still very imperfect (Bradford). There may be an influence on the systemic blood-pressure without any on the pulmonary, and the pressure in the one may rise while it falls in the other, or it may rise and fall in both together. The researches of Brodie and Dixon indicate that drugs which raise the peripheral blood pressure by vaso-constriction increase the total blood in the lung. Thus ergot, the remedy perhaps most commonly used, causes a distinct rise in the pulmonary blood-pressure, while aconite produces a definite fall.

The question is beset with difficulties, and experimental work is by no means in accord. In a recent study Wiggers concludes that in the early stages of hæmoptysis, when the breathing is not altered, lowering of the blood pressure within the pulmonary circuit can not be accomplished by the nitrites, but this can only be done with the cardiac depressants, such as chloroform and the pituitary extracts; and in the later stages of an attack, when the heart is very rapid, pituitary extract is the only drug that raises systemic pressure while simultaneously lowering that in the pulmonary circuit.

The anatomical condition in hæmoptysis is either hyperæmia of the bronchial mucosa (or of the lung tissue) or a perforated vessel. In the latter case the patient often passes rapidly beyond treatment, though there are instances of the most profuse hæmorrhage, which must have come from a perforated artery or a ruptured aneurism, in which recovery has occurred. Practically, for treatment, we should separate these cases, as the remedies which would be applicable in the case of congested and bleeding mucosa would be as much out of place in a case of hæmorrhage from ruptured aneurism as in a cut radial artery. When the blood is brought up in large quantities, it is almost certain either that an aneurism has ruptured or a vessel has been eroded. In the instances in which the sputum is blood tinged or when the blood is in smaller quantities, bleeding comes by diapedesis from hyperæmic vessels. In such cases the hæmorrhage may be beneficial in relieving the congested blood-vessels.

The indications are to reduce the frequency of the heart-beats and to lower the blood-pressure. The truth, *Das Blut ist ein ganz besonderer Saft,* is

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strikingly emphasized by the frightened state of the patient. Rest of the body and peace of the mind—*"quies, securitas, silentium"* of Celsus—should be secured. If there is marked restlessness, morphia hypodermically (gr. ⅙, 0.011 gm.) is advisable. Turn the patient on the affected side, if known, as the regurgitation is less apt to occur into the bronchi of the sound lung. As Aretæus remarks, in hæmoptysis the patient despairs from the first, and needs to be strongly reassured. Death is rarely due directly to hæmoptysis; patients die after, not of it (S. West). In the majority of cases of mild hæmoptysis this is sufficient. Even when the patient insists upon going about, the bleeding may stop spontaneously. The diet should be light and unstimulating. Alcohol should not be used. The patient may, if he wishes, have ice to suck. Small doses of aromatic sulphuric acid may be given, but unless the bleeding is protracted styptic and astringent medicines are not indicated. For cough, which is always present and disturbing, opium should be freely given, and is of all medicines most serviceable in hæmoptysis. Digitalis should not be used, as it raises the blood-pressure in the pulmonary artery. Aconite, as it lowers the pressure, may be used when there is much vascular excitement. Ergot, tannic acid, and lead, which are so much employed, have little or no influence in hæmoptysis; ergot probably does harm. One of the most satisfactory means of lowering the blood-pressure is purgation, and when the bleeding is protracted salts may be freely given. In profuse hæmoptysis, such as comes from erosion of an artery or the rupture of an aneurism, a fatal result is common, and yet post mortem evidence shows that thrombosis may occur with healing in a rupture of considerable size. The fainting induced by the loss of blood is probably the most efficient means of promoting thrombosis, and it was on this principle that formerly patients were bled from the arm, or from both arms, as in the case of Laurence Sterne. Ligatures, or Esmarch's bandages, placed around the legs may serve temporarily to check the bleeding. The ice-bag ~~on the sternum~~ is of doubtful utility. In a protracted case, Cayley induced pneumothorax, ~~but without effect~~.

Briefly, then, we may say that hæmorrhage from rupture of aneurism or erosion of a blood-vessel usually proves fatal. The fainting induced by the loss of blood is beneficial, and, if the patient can be kept alive for twenty-four hours, a thrombus of sufficient strength to prevent further bleeding may form. The chief danger is the inundation of the bronchial system with the blood, so that while the hæmorrhage is profuse the cough should be encouraged. Opium should not then be used, and stimulants should be given with caution.

In the other group, in which the hæmorrhage comes from a congested area and is limited, the patient gets well if kept absolutely quiet, and fatal hæmorrhage probably never occurs from this source. Rest, reduction of the blood-pressure by minimum diet, purging, if necessary, and the administration of opium to allay the cough are the main indications.

II. CHRONIC INTERSTITIAL PNEUMONIA

~~(*Cirrhosis of the Lung*—*Fibroid Phthisis*)~~

A fibroid change may have its starting point in the tissue about the bronchi and blood-vessels, the interlobular septa, the alveolar walls, or in the

pleura. So diverse are the forms and so varied the conditions under which this change occurs that a proper classification is extremely difficult. We may recognize, however, two chief forms—the *local*, involving only a limited area of the lung substance, and the *diffuse*, invading either both lungs or an entire organ.

Etiology.—(*a*) LOCAL fibroid change in the lungs is common. It is a constant accompaniment of tubercle, in the evolution of which interstitial changes play a very important rôle. In tumors, abscess, gummata, hydatids, and emphysema it also occurs. Fibroid processes are frequently met with at the apices of the lung and may be due either to a limited healed tuberculosis, to fibroid induration in consequence of pigment, or, in a few instances, may result from thickening of the pleura.

(*b*) DIFFUSE INTERSTITIAL PNEUMONIA is met with: (1) As a sequence of *acute fibrinous pneumonia*. Although extremely rare, this is recognized as a possible termination. From unknown causes resolution fails to take place. Organization goes on in the fibrinous plugs within the air-cells and the alveolar walls become greatly thickened by a new growth, first of nuclear and subsequently of fibrillated connective tissue. Macroscopically there is produced a smooth, grayish, homogeneous tissue which has the peculiar translucency of all new-formed connective tissue. This has been called gray induration. A majority of the cases terminate within a few months, but instances which have been followed from the outset are very rare.

(2) *Chronic Broncho-pneumonia*.—The relation of broncho-pneumonia to cirrhosis of the lung has been specially studied by Charcot, who states that it may follow the acute or subacute form of this disease, particularly in children. The fibrosis extends from the bronchi, which are usually found dilated. Bronchiectasis itself may be followed by fibrosis of the lung. The alveolar walls are thickened and the lobules converted into firm grayish masses, in which there is no trace of normal lung tissue. This process may go on and involve an entire lobe or even the whole lung. Many of these cases are tuberculous from the outset.

(3) *Pleurogenous Interstitial Pneumonia*.—Charcot applies this term to that form of cirrhosis of the lung which follows invasion from the pleura. Doubt has been expressed by some writers whether this really occurs. While Wilson Fox was probably correct in questioning whether an entire lung can become cirrhosed by the gradual invasion from the pleura, there can be no doubt that there are instances of primitive dry pleurisy, which, as Sir Andrew Clark has pointed out, gradually compresses the lung and at the same time leads to interstitial cirrhosis. This may be due in part to the fibroid change which follows prolonged compression. In some cases there seems to be a distinct connection between the greatly thickened pleura and the dense strands of fibrous tissue passing from it into the lung substance. Instances occur in which one lobe or the greater part of it presents, on section, a mottled appearance, owing to the increased thickness of the interlobar septa—a condition which may exist without a trace of involvement of the pleura. In many other cases, however, the extension seems to be so definitely associated with pleurisy that there is no doubt as to the causal connection, between the two processes. In these instances the lung is removed with great difficulty, owing to the thickness and close adhesion of the pleura to the chest wall.

(4) *Chronic interstitial pneumonia*, due to inhalation of dust, which is considered in a separate section.

(5) *Syphilis* of the lung may present the features of a chronic fibrosis.

(6) Indurative changes in the lung may follow the compression by aneurism or new growth or the irritation of a foreign body in a bronchus.

Morbid Anatomy.—There are two chief forms, the massive or lobar and the insular or broncho-pneumonic form. In the massive type the disease is unilateral; the chest of the affected side is sunken, deformed, and the shoulder much depressed. On opening the thorax the heart is seen drawn far over to the affected side. The unaffected lung is emphysematous and covers the greater portion of the mediastinum. It is scarcely credible in how small a space, close to the spine, the cirrhosed lung may lie. The adhesions between the pleural membranes may be extremely dense and thick, particularly in the pleurogenous cases; but when the disease has originated in the lung there may be little thickening of the pleura. The organ is airless, firm, and hard. It strongly resists cutting, and on section shows a grayish fibroid tissue of variable amount, through which pass the blood-vessels and bronchi. The latter may be either slightly or enormously dilated. There are instances in which the entire lung is converted into a series of bronchiectatic cavities and the cirrhosis is apparent only in certain areas or at the root. The tuberculous cases can usually be differentiated by the presence of an apical cavity, not bronchiectatic, often large, and the other lung almost invariably shows tuberculous lesions. Aneurisms of the pulmonary artery are not infrequent in the cavities. The other lung is always greatly enlarged and emphysematous. The heart is hypertrophied, particularly the right ventricle, and there may be marked atheromatous changes in the vessels. An amyloid condition of the viscera is found in some cases.

In the broncho-pneumonic form the areas are smaller, often centrally placed, and most frequently in the lower lobes. They are deeply pigmented, show dilated bronchi, and when multiple are separated by emphysematous lung tissue.

A *reticular form* of fibrosis of the lung has been described by Percy Kidd and W. McCollum, in which the lungs are intersected by grayish fibroid strands following the lines of the interlobular septa.

Symptoms and Course.—The disease is essentially chronic, extending over a period of many years, and when once the condition is established the health may be fairly good. In a well marked case the patient complains only of his chronic cough, perhaps a slight shortness of breath. In other respects he is quite well, and is usually able to do light work. The cases are commonly regarded as phthisical, though there may be scarcely a symptom of that affection except the cough. There are instances, however, of fibroid phthisis which can not be distinguished from cirrhosis of the lung except by the presence of tubercle bacilli in the expectoration. As the bronchi are usually dilated, the symptoms and physical signs may be those of bronchiectasis. The cough is paroxysmal and the expectoration is generally copious and of a muco-purulent or sero-purulent nature. It is sometimes fetid. Hæmorrhage is by no means infrequent, and occurred in more than one-half of the cases analyzed by Bastian. Walking on the level and in the ordinary affairs of life, the patient

may show no shortness of breath, but in the ascent of stairs and on exertion there may be dyspnœa.

Physical Signs.—*Inspection.*—The affected side of the chest is immobile, retracted, and shrunken, and contrasts in a striking way with the voluminous healthy one. The intercostal spaces are obliterated and the ribs may even overlap. The shoulder is drawn down and from behind it is seen that the spine is bowed. The muscles of the shoulder-girdle are wasted. The heart is greatly displaced, being drawn over by the shrinkage of the lung to the affected side. When the left lung is affected there may be a large area of visible impulse in the second, third, and fourth interspaces. Mensuration shows a great diminution in the affected side, and with the saddle-tape the expansion may be seen to be negative. The *percussion* note varies with the condition of the bronchi. It may be absolutely flat, particularly at the base or at the apex. In the axilla there may be a flat tympany or even an amphoric note over a large sacculated bronchus. On the opposite side the percussion note is usually hyperresonant. On auscultation the breath-sounds have either a cavernous or amphoric quality at the apex, and at the base are feeble, with mucous, bubbling râles. The voice-sounds are usually exaggerated. Cardiac murmurs are not uncommon, particularly late in the disease, when the right heart fails. These are, of course, the physical signs of the disease when it is well established. They naturally vary considerably, according to the stage of the process. The disease is essentially chronic, and may persist for fifteen or twenty years. Death occurs sometimes from hæmorrhage, more commonly from gradual failure of the right heart with dropsy, and occasionally from amyloid degeneration of the organs.

Diagnosis.—The diagnosis is never difficult. It may be impossible to say, without a clear history, whether the origin is pleuritic or pneumonic. Between cases of this kind and fibroid phthisis it is not always easy to discriminate, as the conditions may be almost identical. When tuberculosis is present, however, even in long-standing cases, bacilli are usually present in the sputum, and there may be signs of disease in the other lung.

Treatment.—It is only for an intercurrent affection or for an aggravation of the cough that the patient seeks relief. Nothing can be done for the condition itself. When possible the patient should live in a mild climate, and avoid exposure to cold and damp. A distressing feature in some cases is the putrefaction of the contents of the dilated tubes, for which the same measures may be used as in fetid bronchitis.

III. PNEUMONOKONIOSIS

Definition.—Under this term, introduced by Zenker, are embraced those forms of fibrosis of the lung due to the inhalation of dusts in various occupations. They have received various names, according to the nature of the inhaled particles—*anthracosis,* or coal-miner's disease; *siderosis,* due to the inhalation of metallic dusts, particularly iron; *chalicosis* and *silicosis,* due to the inhalation of mineral dusts, producing the so-called stone-cutter's phthisis, or the "grinder's rot" of the Sheffield workers.

Etiology.—The dust particles inhaled into the lungs are dealt with exten-

tuberculosis!

Dust does not favor tuberculosis because caseo-fibrosis
which is often due to tubes.

organic dust has no effect in causing disease.

Dusty trades.. The majority show no change for it has
escaped ab ove effect.

—

Dysph. in tubes. often due to diaphragm fixed adhesions.

sively by the ciliated epithelium and by the phagocytes, which exist normally in the respiratory organs. The ordinary mucous corpuscles take in a large number of the particles, which fall upon the trachea and main bronchi. The cilia sweep the mucus out to a point from which it can be expelled by coughing. It is doubtful if the particles ever reach the air-cells, but the swollen alveolar cells (in which they are in numbers) probably pick them up on the way. The mucous and the alveolar cells are the normal respiratory scavengers. In dwellers in the country, ~~in which~~ the air is pure, they are able to prevent the access of dust particles to the lung tissue, so that even in adults these organs present a rosy tint, very different from the dark, carbonized appearance of the lungs of dwellers in cities. When the impurities in the air are very abundant, a certain proportion of the dust particles escapes these cells and penetrates the mucosa, reaching the lymph spaces, where they are attacked at once by the cells of the connective-tissue stroma, which are capable of ingesting and retaining a large quantity. In coal-miners, coal-heavers, and others whose occupations necessitate the constant breathing of a very dusty atmosphere even these forces are insufficient. Vansteenberghe and Grysez ~~have~~ demonstrated that pulmonary anthracosis may be induced by passing an emulsion of china ink into the stomach of an animal through a catheter. From a long series of experiments they conclude that anthracosis is due to the intestinal absorption of carbon particles arrested in the nose and pharynx, and then swallowed. Their experiments further show that both the tracheal and intestinal routes are used—through the former the particles reach the bronchi and external portions of the alveoli, through the latter the parenchyma of the lung. Occasionally in anthracosis the carbon grains reach the general circulation, and the coal dust is found in the liver and spleen. As Weigert ~~has~~ shown, this occurs when the densely pigmented bronchial glands closely adhere to the pulmonary veins, through the walls of which the carbon particles pass to the general circulation. The lung tissue has a remarkable tolerance for these particles; but by constant exposure a limit is reached, and ~~there is brought about~~ a ~~very~~ definite pathological condition, an interstitial sclerosis. In coal-miners this may occur in patches, even before the lung tissue is uniformly infiltrated with the dust. In others it appears only after the entire organs have become so laden that they are dark in color, and an ink-like juice flows from the cut surface. The lungs of a miner may be black throughout and yet show no local lesions and be everywhere crepitant.

Morbid Anatomy.—The particles of carbon are found deposited in large numbers in the follicular cords of the tracheal and bronchial glands and of the peri-bronchial and peri-arterial lymph nodules, and in these they finally excite proliferation of the connective tissue elements. It is by no means uncommon to find in persons whose lungs are only moderately carbonized the bronchial glands sclerosed and hard. In anthracosis the fibroid changes usually begin in the peri-bronchial lymph tissue, and in the early stage of the process the sclerosis may be largely confined to these regions. A Nova Scotian miner, aged thirty-six, died ~~under my care,~~ at the Montreal General Hospital, of black small-pox, after an illness of a few days. In his lungs (externally coal-black) there were round and linear patches ranging in size from a pea to a hazel-nut, of an intensely black color, airless and firm, and surrounded by a crepitant tissue, slate gray in color. In the centre of each of these areas

was a small bronchus. Many of them were situated just beneath the pleura, and formed typical examples of limited fibroid broncho-pneumonia. In addition there is usually thickening of the alveolar walls, particularly in certain areas. By the gradual coalescence of these fibroid patches large portions of the lung may be converted into firm areas of cirrhosis, grayish black in the case of the coal-miner, steel gray in the case of the stone-worker. In the case of a Cornish miner, aged sixty-three, who died under my care, one of these fibroid areas measured 18 by 6 cm. and 4.5 cm. in depth.

A second important factor in these cases is chronic bronchitis, which is present in a large proportion and really causes the chief symptoms. A third is the occurrence of emphysema, which is almost invariably associated with long-standing cases of pneumonokoniosis. With the changes so far described, unless the cirrhotic area is unusually extensive, the case may present the features of chronic bronchitis with emphysema, but finally another element comes into play. In the fibroid areas softening occurs, probably a process of necrosis similar to that by which softening is produced in fibro-myomata of the uterus. At first these are small and contain a dark liquid. Charcot calls them *ulcères du poumon*. They rarely attain a large size unless a communication is formed with the bronchus, in which case they may become converted into suppurating cavities.

Anthracosis and Tuberculosis.—In the Pennsylvania anthracite district tuberculosis is relatively less common among the miners, the figures for ten years at Scranton for male adults being 3.37 per cent. in mine workers, 9.97 per cent. in those of other occupations (Wainwright). Goldman in Germany, Oliver and Trotter in England, all agree upon the comparative rarity of tuberculosis among coal miners. Though this may be attributed in part to the improved ventilation of the mines, it has also probably something to do with the less favorable soil offered to the bacilli in a lung infiltrated with coal dust.

The *siderosis* induced by the oxide of iron causes an interstitial pneumonia similar to anthracosis. Workers in brass and in bronze are liable to a like affection.

Chalicosis and *silicosis*, due to the deposit of particles of silex and alumina, are found in the makers of mill-stones, particularly the French mill-stones, and also in knife and axe grinders and stone-cutters. It prevails extensively among the Rand miners of South Africa (gold-miners' phthisis), and in the workers of the Australian and Tasmanian mines. Anatomically, this form is characterized by the production of nodules of various sizes, which are cut with the greatest difficulty and sometimes present a curious grayish, even glittering, crystalloid appearance.

Workers in flax and in cotton, and grain-shovellers are also subject to these chronic interstitial changes in the lungs.

Symptoms.—The symptoms do not come on until the patient has worked for a variable number of years in the dusty atmosphere. As a rule there are cough and failing health for a prolonged period of time before complete disability. The coincident emphysema is responsible in great part for the shortness of breath and wheezy condition of these patients. The expectoration is usually muco-purulent, often profuse, and in anthracosis very dark in color—

the so-called "black spit," while in chalicosis there may be seen under the microscope the bright angular particles of silica.

Even when there are physical signs of cavity, tubercle bacilli are not necessarily, and indeed in my experience are not usually present. It is remarkable for how long a time a coal-miner may continue to bring up sputum laden with coal particles even when there are signs only of a chronic bronchitis. Many of the particles are contained in the cells of the alveolar epithelium. In these instances it appears that an attempt is made by the leucocytes to rid the lungs of some of the carbon grains.

Diagnosis.—The diagnosis of the condition is rarely difficult; the expectoration is usually characteristic. It must always be borne in mind that chronic bronchitis and emphysema form essential parts of the process and that in late stages there may be tuberculous infection.

Prophylaxis.—Much has been done to reduce the prevalence of the disease in England by proper ventilation of works and the protection of the men. The conversion of dry into wet mining prevents the distribution of the injurious dust.

Treatment.—The treatment of the condition is practically that of chronic bronchitis and emphysema.

IV. EMPHYSEMA

Definition.—The condition in which the infundibular passages and the alveoli are dilated and the alveolar walls atrophied.

Floyer of Litchfield first described the anatomical condition and spoke of the disease as "flatulent asthma" (1698), meaning a disorder in which the lungs were blown up with air.

A practical division may be made into compensatory, hypertrophic, and atrophic forms, the acute vesicular emphysema, and the interstitial forms. The last two do not in reality come under the above definition, but for convenience they may be considered here.

1. COMPENSATORY EMPHYSEMA

Whenever a region of the lung does not expand fully in inspiration, either another portion of the lung must expand or the chest wall sink in order to occupy the space. The former almost invariably occurs. We have already mentioned that in broncho-pneumonia there is a vicarious distention of the air-vesicles in the adjacent healthy lobules, and the same happens in the neighborhood of tuberculous areas and cicatrices. In general pleural adhesions there is often compensatory emphysema, particularly at the anterior margins of the lung. The most advanced example of this form is seen in cirrhosis, when the unaffected lung increases greatly in size, owing to distention of the air-vesicles. A similar though less marked condition is seen in extensive pleurisy with effusion and in pneumothorax.

At first, this distention of the air-vesicles is a simple physiological process and the alveolar walls are stretched but not atrophied. Ultimately, however, in many cases they waste and the contiguous air-cells fuse, producing true emphysema.

2. HYPERTROPHIC EMPHYSEMA

The large-lunged emphysema of Jenner, also known as substantive or idiopathic emphysema, is a well-marked clinical affection, characterized by enlargement of the lungs, due to distention of the air-cells and atrophy of their walls, and clinically by imperfect aëration of the blood and more or less marked dyspnœa.

Etiology.—Emphysema is the result of persistently high intra-alveolar tension acting upon a congenitally weak lung tissue. Strongly in favor of the view that the nutritive change in the air-cells is the primary factor are the markedly hereditary character of the disease and the frequency with which it starts early in life. To James Jackson, Jr., of Boston, we owe the first observations on the hereditary character of emphysema. Working under Louis' direction, he found that in 18 out of 28 cases one or both parents were affected.

In childhood it may follow recurring asthmatic attacks due to adenoid vegetations. It may occur, too, in several members of the same family. We are still ignorant as to the nature of this congenital pulmonary weakness. Cohnheim thinks it probably due to a defect in the development of the elastic-tissue fibres—a statement which is borne out by Eppinger's observations.

Heightened pressure within the air-cells may be due to forcible inspiration or expiration. Much discussion has taken place as to the part played by these two acts in the production of the disease. The inspiratory theory was advanced by Laennec and subsequently modified by Gairdner, who held that in chronic bronchitis areas of collapse were induced, and compensatory distention took place in the adjacent lobules. This unquestionably does occur in the vicarious or compensatory emphysema, but it probably is not a factor of much moment in the form now under consideration. The expiratory theory, which was supported by Mendelssohn and Jenner, accounts for the condition in a much more satisfactory way. In all straining efforts and violent attacks of coughing the glottis is closed and the chest walls are strongly compressed by muscular efforts, so that the strain is thrown upon those parts of the lung least protected, as the apices and the anterior margins, in which we always find the emphysema most advanced. The sternum and costal cartilages gradually yield to the heightened intrathoracic pressure and are, in advanced cases, pushed forward, giving the characteristic rotundity to the thorax.

Freund's Theory.—A primary disease of the costal cartilages—a chronic hyperplasia with premature ossification is believed to bring about gradually a state of rigid dilatation of the chest, to which the emphysema is secondary. Recent observations make it probable that there is a group of cases in which such changes occur in young persons, particularly in the cartilages of the first three ribs. Niemeyer says that he had met with a few such cases, and there have been reported recently instances in which the cartilages increased in size and stood out prominently. For such a condition what is now called Freund's operation (of resection) would be indicated.

Of other etiological factors occupation is the most important. The disease is met with in players on wind instruments, in glass-blowers, and in occupations necessitating heavy lifting or straining. Whooping-cough and

bronchitis play an important rôle, not so much in the changes which they induce in the bronchi as in consequence of the prolonged attacks of coughing.

Morbid Anatomy.—The thorax is capacious, usually barrel-shaped, and the cartilages are calcified. On removal of the sternum, the anterior mediastinum is found completely occupied by the margins of the lungs, and the pericardial sac may not be visible. The organs are very large and have lost their elasticity, so that they do not collapse either in the thorax or when placed on the table. The pleura is pale and there is often an absence of pigment, sometimes in patches, termed by Virchow *albinism* of the lung. To the touch they have a peculiar, downy, feathery feel, and pit readily on pressure. This is one of the most marked features. Beneath the pleura greatly enlarged air-vesicles may be readily seen. They vary in size from .5 to 3 mm., and irregular bullæ, the size of a walnut or larger, may project from the free margins. The best idea of the extreme rarefaction of the tissue is obtained from sections of a lung distended and dried. At the anterior margins the structure may form an irregular series of air-chambers, resembling the frog's lung. On careful inspection with the hand-lens, remnants of the interlobular septa or even of the alveoli may be seen on these large emphysematous vesicles. Though general, the distention is more marked, as a rule, at the anterior margins, and is often specially marked at the inner surface of the lobe near the root, where in extreme cases air-spaces as large as a hen's egg may sometimes be found. Microscopically there is seen atrophy of the alveolar walls, by which is produced a coalescence of neighboring air-cells. In this process the capillary network disappears before the walls are completely atrophied. The loss of the elastic tissue is a special feature. It is stated, indeed, that in certain cases there is a congenital defect in the development of this tissue. The epithelium of the air-cells undergoes a fatty change, but the large distended air-spaces retain a pavement layer.

The bronchi show important changes. In the larger tubes the mucous membrane may be rough and thickened from chronic bronchitis; often the longitudinal lines of submucous elastic tissue stand out prominently. In the advanced cases many of the smaller tubes are dilated, particularly when, in addition to emphysema, there are peri-bronchial fibroid changes. Bronchiectasis is not, however, an invariable accompaniment of emphysema, but, as Laennec remarks, it is difficult to understand why it is not more common. Of associated morbid changes the most important are found in the heart. The right chambers are dilated and hypertrophied, the tricuspid orifice is large, and the valve segments are often thickened at the edges. In advanced cases the cardiac hypertrophy is general. The pulmonary artery and its branches may be wide and show marked atheromatous changes.

The changes in the other organs are those commonly associated with prolonged venous congestion. Pneumothorax may follow the rupture of an emphysematous bleb.

Symptoms.—The disease may be tolerably advanced before any special symptoms occur. A child, for instance, may be somewhat short of breath on going up-stairs or may be unable to run and play as other children without great discomfort; or, perhaps, has attacks of slight lividity. Doubtless much depends upon the completeness of cardiac compensation. When this is perfect, there may be no special interruption of the pulmonary circulation and,

except with violent exertion, there is no interference with the aëration of the blood. In well-marked cases the following are the most important symptoms: *Dyspnœa*, which may be felt only on slight exertion, or may be persistent, and aggravated by intercurrent attacks of bronchitis. The respirations are often harsh and wheezy, and expiration is distinctly prolonged.

Cyanosis of an extreme grade is more common in emphysema than in other affections with the exception of congenital heart-disease. ~~So far as I know it is the only~~ disease in which a patient may be able to go about and even to walk into the hospital or consulting-room with a lividity of startling intensity. The contrast between the extreme cyanosis and the comparative comfort of the patient is very striking. In other affections of the heart and lungs associated with a similar degree of cyanosis the patient is invariably in bed and usually in a state of orthopnœa. One condition must be ~~here~~ referred to, viz., the extraordinary cyanosis in cases of poisoning by aniline products, ~~which is~~ in most part due to the conversion of the hæmoglobin into methæmoglobin.

Bronchitis with associated cough is a frequent symptom and often the direct cause of the pulmonary distress. The contrast between emphysematous patients in the winter and summer is marked in this respect. In the latter they may be comfortable and able to attend to their work, but with the cold and changeable weather they are laid up with attacks of bronchitis. Finally, in fact, the two conditions become inseparable and the patient has persistently more or less cough. The acute bronchitis may produce attacks not unlike asthma. In some instances this is true spasmodic asthma, with which emphysema is frequently associated.

As age advances, and with successive attacks of bronchitis, the condition grows slowly worse. In hospital practice it is common to admit patients over sixty with well marked signs of advanced emphysema. The affection can generally be told at a glance—the rounded shoulders, barrel chest, the thin yet oftentimes muscular form, and sometimes, ~~I think~~, a very characteristic facial expression.

There is another group of younger patients from twenty-five to forty years of age who, winter after winter, have attacks of intense cyanosis in consequence of an aggravated bronchial catarrh. On inquiry we find that these patients have been short-breathed from infancy, and they belong to a category in which there has been a primary defect of structure in the lung tissue.

Physical Signs.—*Inspection.*—The thorax is markedly altered in shape; the antero-posterior diameter is increased and may be even greater than the lateral, so that the chest is barrel-shaped. The appearance is somewhat as if the chest was in a permanent inspiratory position. The sternum and costal cartilages are prominent. The lower zone of the thorax looks large and the intercostal spaces are much widened, particularly in the hypochondriac regions. The sternal fossa is deep, the clavicles stand out with great prominence, and the neck looks shortened from the elevation of the thorax and the sternum. A zone of dilated venules may be seen along the line of attachment of the diaphragm. Though this is common in emphysema, it is by no means peculiar to it or indeed to any special affection.

The curve of the spine is increased and the back is remarkably rounded, so that the scapulæ seem to be almost horizontal. Mensuration shows the

In children - sometimes disappearing of expiration - lower edge of lung found below usual level - Pneumonia or rib sounds - Breathing exercises - to train expiration - air

rounded form of the chest and the very slight expansion on deep inspiration. The respiratory movements, which may look energetic and forcible, exercise little or no influence. The chest does not expand, but there is a general elevation. The inspiratory effort is short and quick; the expiratory movement is prolonged. There may be retraction instead of distention in the upper abdominal region during inspiration, and there is sometimes seen a transverse curve crossing the abdomen at the level of the twelfth rib. The apex beat of the heart is not visible, and there is usually marked pulsation in the epigastric region. The cervical veins stand out prominently and may pulsate.

Palpation.—The vocal fremitus is somewhat enfeebled but not lost. The apex beat can rarely be felt. There is a marked shock in the lower sternal region and very distinct pulsation in the epigastrium. *Percussion* gives greatly increased resonance, full and drum-like—what is sometimes called hyperresonance. The note is not often distinctly tympanitic in quality. The percussion note is greatly extended, the heart dulness may be obliterated, the upper limit of liver dulness is greatly lowered, and the resonance may extend to the costal margin. Behind, a clear percussion note extends to a much lower level than normal. The level of splenic dulness, too, may be lowered.

On *auscultation* the breath-sounds are usually enfeebled and may be masked by bronchitic râles. The most characteristic feature is the prolongation of the expiration, and the normal ratio may be reversed, 4 to 1 instead of 1 to 4. It is often wheezy and harsh and associated with coarse râles and sibilant rhonchi. It is said that in interstitial emphysema there may be a friction sound heard, not unlike that of pleurisy. The heart-sounds are usually feeble but clear; in advanced cases, when there is marked cyanosis, a tricuspid regurgitant murmur may be heard. Accentuation of the pulmonary second sound may be present.

Course.—The course of the disease is slow but progressive, the recurring attacks of bronchitis aggravating the condition. Death may occur from intercurrent pneumonia, either lobar or lobular, and dropsy may supervene from cardiac failure. Occasionally death results from overdistention of the heart, with extreme cyanosis. Duckworth has called attention to the occasional occurrence of fatal hæmorrhage in emphysema. In an old emphysematous patient at the Montreal General Hospital death followed the erosion of a main branch of the pulmonary artery by an ulcer near the bifurcation of the trachea.

Treatment.—Practically, the measures mentioned in connection with bronchitis should be employed. In children with asthma and emphysema the nose should be carefully examined. No remedy is known which has any influence over the progress of the condition itself. Bronchitis is the great danger of these patients, and therefore when possible they should live in an equable climate. They do well in southern California and in Egypt. In consequence of the venous engorgement they are liable to gastric and intestinal disturbance, and it is particularly important to keep the bowels regulated and to avoid flatulency, which often seriously aggravates the dyspnœa. Patients who come into the hospital in a state of urgent dyspnœa and lividity, with great engorgement of the veins, particularly if they are young and vigorous, should be bled freely. Inhalation of oxygen may be used. Strychnine will be found specially useful, and breathing exercises are sometimes helpful. Breathing

of compressed air in a pneumatic cabinet gives temporary relief. Resection of the first costal cartilage or of the first three cartilages on either side has been practiced (Freund's operation). It is not likely to be of any benefit in the aged in whom the condition is established, but in a special group in the young in which the primary trouble appears to be in the cartilages—what has been called *Freund's Disease*—the operation may be practiced and good results have followed.

3. ATROPHIC EMPHYSEMA

A senile change, called by Sir William Jenner small-lunged emphysema, is really a primary atrophy of the lung, coming on in advanced life, and scarcely constitutes a special affection. It occurs in "withered-looking old persons" who may perhaps have had a winter cough and shortness of breath for years. In striking contrast to the essential hypertrophic emphysema, the chest is small and the ribs obliquely placed. The thoracic muscles are usually atrophied. The lung presents a remarkable appearance, being converted into a series of large vesicles, on the walls of which the remnants of air-cells may be seen.

4. ACUTE VESICULAR EMPHYSEMA

When death occurs from bronchitis of the smaller tubes, when strong inspiratory efforts have been made, the lungs are large in volume and the air-cells are much distended. Clinically, this condition may occur rapidly in cases of cardiac asthma and angina pectoris. The area of pulmonary resonance is much increased, and on auscultation there are heard everywhere piping râles and prolonged expiration. A similar condition may follow pressure on the vagi.

5. INTERSTITIAL EMPHYSEMA

Beads of air are seen in the interlobular and subpleural tissue, sometimes forming large bullæ beneath the pleura. A rare event is rupture close to the root of the lung, and the passage of air along the trachea into the subcutaneous tissues of the neck. After tracheotomy just the reverse may occur and the air may pass from the tracheotomy wound along the windpipe and bronchi and appear beneath the surface of the pleura. From this interstitial emphysema spontaneous pneumothorax may arise in healthy persons.

V. GANGRENE OF THE LUNG

Etiology.—Gangrene of the lung is not an affection *per se*, but occurs in a variety of conditions when necrotic areas undergo putrefaction. It is not easy to say why sphacelus should occur in one case and not in another, as the germs of putrefaction are always in the air-passages, and yet necrotic territories rarely become gangrenous. Total obstruction of a pulmonary artery, as a rule, causes infarction, and the area shut off does not often, though it may, sphacelate. Another factor would seem to be necessary—probably a lowered tissue resistance, the result of general or local causes. It is met with (1) as a sequence of lobar pneumonia. This rarely occurs in a previously

.good vessels may follow /

healthy person—more commonly in the debilitated or in the diabetic subject. (2) Gangrene is very prone to follow the aspiration pneumonia, since the foreign particles rapidly undergo putrefactive changes. Of a similar nature are the cases of gangrene due to perforation of cancer of the œsophagus into the lung or into the bronchus. (3) The putrid contents of a bronchiectatic, more commonly of a tuberculous, cavity may excite gangrene in the neighboring tissues. The pressure bronchiectasis following aneurism or tumor may lead to extensive sloughing. (4) Gangrene may follow simple embolism of the pulmonary artery. More commonly, however, the embolus is derived from a part which is mortified or comes from a focus of bone disease. In typhus and in typhoid fever gangrene of the lung may follow thrombosis of one of the larger branches of the pulmonary artery. A case occurred in my wards in October, 1897, in connection with a typhoid septicæmia. Typhoid bacilli were isolated from the lung. Lastly, gangrene of the lung may occur in conditions of debility during convalescence from protracted fever—occasionally, indeed, without our being able to assign any reasonable cause.

Morbid Anatomy.—Laennec, who first accurately described pulmonary gangrene, recognized a diffuse and a circumscribed form. The former, though rare, is sometimes seen in connection with pneumonia, more rarely after obliteration of a large branch of the pulmonary artery. It may involve the greater part of a lobe, and the lung tissue is converted into a horribly offensive greenish-black mass, torn and ragged in the centre. In the circumscribed form there is well-marked limitation between the gangrenous area and the surrounding tissue. The focus may be single or there may be two or more. The lower lobe is more commonly affected than the upper, and the peripheral more than the central portion of the lung. A gangrenous area is at first uniformly greenish brown in color; but softening rapidly takes place with the formation of a cavity with shreddy, irregular walls and a greenish, offensive fluid. The lung tissue in the immediate neighborhood shows a zone of deep congestion, often consolidation, and outside this an intense œdema. In the embolic cases the plugged artery can sometimes be found. When rapidly extending, vessels may be opened and a copious hæmorrhage ensue. Perforation of the pleura is not uncommon. The irritating decomposing material usually excites the most intense bronchitis. Embolic processes are not infrequent. There is a remarkable association in some cases between circumscribed gangrene of the lung and abscess of the brain. It has been referred to under the section on bronchiectasis.

Symptoms and Course.—Usually definite symptoms of local pulmonary disease precede the characteristic features of gangrene. These, of course, are very varied, depending on the nature of the trouble. The sputum is very characteristic. It is intensely fetid—usually profuse—and, if expectorated into a conical glass, separates into three layers—a greenish brown, heavy sediment; an intervening thin liquid, which sometimes has a greenish or a brownish tint; and, on top, a thick, frothy layer. Spread on a glass plate, the shreddy *débris* of lung tissue can readily be picked out. Even large fragments of lung may be coughed up. Robertson, of Onancock, Va., sent me one several centimetres in length, which had been expectorated by a lad of eighteen, who had severe gangrene and recovered. Microscopically, elastic fibres are found in abundance, with granular matter, pigment grains, fatty crystals, bacteria, and

leptothrix. It is stated that elastic tissue is sometimes absent, but I have never met with such an instance. The peculiar plugs of sputum which occur in bronchiectasis are not found. Blood is often present, and, as a rule, is much altered. The sputum has, in a majority of the cases, an intensely fetid odor, which is communicated to the breath and may permeate the entire room. It is much more offensive than in fetid bronchitis or in abscess of the lung. The fetor is particularly marked when there is free communication between the gangrenous cavities and the bronchi. On several occasions I have found, post mortem, localized gangrene, which had been unsuspected during life, and in which there had been no fetor of the breath.

The physical signs, when extensive destruction has occurred, are those of cavity, but the limited circumscribed areas may be difficult to detect. Bronchitis is always present. The X-ray examination may aid in diagnosis.

Among the general symptoms may be mentioned fever, usually of moderate grade; the pulse is rapid, and very often the constitutional depression is severe. But the only special features indicative of gangrene are the sputum and the fetor of the breath. The patient generally sinks from exhaustion. Fatal hæmorrhage may ensue.

Treatment.—The treatment of gangrene is very unsatisfactory. The indications, of course, are to disinfect the gangrenous area, but this is often impossible. An antiseptic spray of carbolic acid may be employed. A good plan is for the patient to use over the mouth and nose an inhaler, which may be charged with a solution of carbolic acid or with guaiacol; the latter drug has also been used hypodermically, with, it is said, happy results in removing the odor. If the signs of cavity are distinct an attempt should be made to cleanse it by direct injections of an antiseptic solution. If the patient's condition is good and the gangrenous region can be localized, surgical interference may be indicated. Successful cases have been reported. The general condition of the patient is always such as to demand the greatest care in the matter of diet and nursing.

VI. ABSCESS OF THE LUNG

Etiology.—Suppuration occurs in the lung under the following conditions: (1) As a sequence of inflammation, either lobar or lobular. Apart from the purulent infiltration this is unquestionably rare, and even in lobar pneumonia the abscesses are of small size and usually involve, as Addison remarked, several points at the same time. On the other hand, abscess formation is extremely frequent in the deglutition and aspiration forms of lobular pneumonia. After wounds of the neck or operations upon the throat, in suppurative disease of the nose or larynx, occasionally even of the ear (Volkmann), infective particles reach the bronchial tubes by aspiration and excite an intense inflammation which often ends in abscess. Cancer of the œsophagus, perforating the root of the lung or into the bronchi, may produce extensive suppuration. The abscesses vary in size from a walnut to an orange, and have ragged and irregular walls, and purulent, sometimes necrotic, contents.

(2) Embolic, so-called metastatic, abscesses, the result of infective emboli, are extremely common in pyæmia. They may be numerous and present very

Signs- abscess. ① dulness. may be or not other signs

① signs on x ray exam.

③ elastic tissue with alveolar arrangement in sputum

Tr. put. lie with affected side uppermost for certain period.

elevation of foot of bed — morphine.

turn head over bed during paroxysms of cough.

Pneumothorax suggested when abscess has communication with bronchus

definite characters. As a rule they are superficial, beneath the pleura, and often wedge-shaped. At first firm, grayish red in color, and surrounded by a zone of intense hyperæmia, suppuration soon follows with the formation of a definite abscess. The pleura is usually covered with greenish lymph, and perforation sometimes takes place with the production of pneumothorax.

(3) Perforation of the lung from without, lodgment of foreign bodies, and, in the right lung, perforation from abscess of the liver or a suppurating echinococcus cyst are occasionally causes of pulmonary abscess.

(4) Suppurative processes play an important part in chronic pulmonary tuberculosis, many of the symptoms of which are due to them.

Symptoms.—Abscess following pneumonia is easily recognized by an aggravation of the general symptoms and by the physical signs of cavity and the characters of the expectoration. Embolic abscesses can not often be recognized, and the local symptoms are generally masked in the general pyæmic manifestations. The character of the sputum is of great importance in determining the presence of abscess. The odor is offensive, yet it rarely has the horrible fetor of gangrene or of putrid bronchitis. In the pus fragments of lung tissue can be seen, and the elastic tissue may be very abundant. The presence of this with the physical signs rarely leaves any question as to the nature of the trouble. Embolic cases usually run a fatal course. Recovery occasionally occurs after pneumonia. In a case following typhoid fever which I saw at the Garfield Hospital Kerr removed two ribs and found free in the pus of a localized empyema a sequestered piece of lung, the size of the palm of the hand, which had sloughed off from the lower lobe. The patient made a good recovery.

Medicinal treatment is of little avail. In abscess of the lung. When well defined and superficial, an attempt should always be made to open and drain it. A number of successful cases have already been treated in this way.

VII. NEW GROWTHS IN THE LUNGS

Etiology and Morbid Anatomy.—While primary tumors are rare, secondary growths are not uncommon. Carcinoma is the most common primary form. Endothelium and sarcoma are less frequently met with.

Varieties.—The following groups may be recognized:

(*a*) ACUTE GALLOPING PLEURO-PNEUMONIC FORM, with a very rapid course—dyspnœa, cough, asphyxia, rapid emaciation and death in from six to twelve weeks. Most of these cases are secondary, sometimes to unrecognized disease elsewhere, but there are instances of the primary disease of this type. It is a remarkable fact that cobalt miners of Schneeberg are very liable to a primary carcinoma of the lung which may run this acute course.

(*b*) CHRONIC PLEURO-PULMONARY CARCINOMA, of which there are several types:

(1) *Broncho-pulmonary Form.*—This, the most typical variety, begins with bronchial symptoms, bloody sputum, loss of weight and strength, and anæmia. The physical signs may be suggestive of tuberculosis, but the earliest indications are usually at the root of the lung. Later there may be cavity formation, with a bronchiectatic type of sputum. Tubercle bacilli are absent

and there may be very large round cells with many fatty granules, representing degenerate cancer cells. The X-ray picture is not distinctive and the cases are usually taken for tuberculosis.

(2) *Mediastinal Type.*—Quite early in this form the glands become involved, increase rapidly, compress the adjoining structures and the type of the disease is that of a mediastinal tumor with its dominant pressure symptoms.

(3) *Pleuritic Type.*—The earliest and dominant symptoms are at the back with pleuritic pain, cough, friction, progressive effusion, and shortness of breath. On tapping, the effusion is usually bloody, though ~~I have known~~ it at first ~~to be~~ clear. In other instances the pleura is early involved with rapid extension, but no effusion. There may be little or no cough, and very slight dyspnœa, with progressive weakness, emaciation, and anæmia as the chief features. Subcutaneous nodules may occur along the ribs, with involvement of both cervical and axillary glands.

From the standpoint of treatment not much is to be expected. The new surgical technique has made the thoracic cavity accessible, and it is ~~quite~~ possible that early explorations may become common in doubtful cases. In a few instances (carcinoma of the lung has been operated upon); in Lenhartz' case the patient remained well for a year, and died two and a half years after operation.

E. DISEASES OF THE PLEURA

I. ACUTE PLEURISY

Anatomically, the cases may be divided into dry or adhesive pleurisy and pleurisy with effusion. Another classification is into primary or secondary forms. According to the course ~~of the disease~~, a division may be made into *acute* and *chronic pleurisy*, and as it is impossible, at present, to group the various forms etiologically, this is perhaps the most satisfactory division. ~~The following forms of acute pleurisy may be considered~~:

1. FIBRINOUS OR PLASTIC PLEURISY

In this the pleural membrane is covered by a sheeting of lymph of variable thickness, which gives it a turbid, granular appearance, or the fibrin may exist in distinct layers. It occurs (1) as an independent affection, following cold or exposure. This form of acute plastic pleurisy without fluid exudate is not common in perfectly healthy individuals. Cases are met with, however, in which the disease sets in with the usual symptoms of pain in the side and slight fever, and there are the physical signs of pleurisy as indicated by the friction. After persisting for a few days, the friction murmur disappears and no exudation occurs. Union takes place between the membranes, and possibly the pleuritic adhesions which are found in such a large percentage of all bodies examined after death originate in these slight fibrinous pleurisies.

Fibrinous pleurisy occurs (2) as a secondary process in acute diseases of the lung, such as pneumonia, which is always accompanied by a certain amount of pleurisy, usually of this form. Cancer, abscess, and gangrene also cause plastic pleurisy when the surface of the lung becomes involved. This

condition is specially associated in a large number of cases with tuberculosis. Pleural pain, stitch in the side, and a dry cough, with marked friction sounds on auscultation, are the initial phenomena in many instances of phthisis. The signs are usually basic.

2. SERO-FIBRINOUS PLEURISY

In a majority of cases of inflammation of the pleura there is, with the fibrin, a variable amount of fluid exudate, which produces the condition known as pleurisy with effusion.

Etiology.—Of 194 cases in fifteen years in my wards, there were 161 males and 33 females. Under twenty years of age there were 20 patients; 18 were over sixty years of age. The greatest number was in the fifth decade, 59. Cold acts as a predisposing agent, which permits the action of various micro-organisms. We have not yet, however, brought all the acute pleurisies into the category of microbic affections, and the fact remains that pleurisy does follow with great rapidity a sudden wetting or a chill. A majority of the cases are tuberculous. This view is based upon: (1) Post mortem evidence. Tubercles have been found in acute cases, thought to have been rheumatic or due to cold. (2) The not infrequent presence of tuberculous lesions, often latent, in the lung or elsewhere. (3) The character of the exudate. If coagulated and the coagulum digested and centrifugalized (Inoscopy), tubercle bacilli are frequently found. Injected into a guinea pig, in amounts of 15 c. c. or more, tuberculosis followed in 62 per cent. (Eichhorst). The cyto-diagnosis shows that as in other tuberculous exudates the mono-nuclear leucocytes predominate. (4) The tuberculin reaction is given in a considerable percentage of the cases. (5) The subsequent history. Of 90 cases of acute pleurisy which had been under the observation of H. I. Bowditch between 1849 and 1879, 32 died of or had phthisis. Among 130 patients with primary pleurisy with effusion, followed for a period of seven years by Hedges, 40 per cent. became tuberculous.

Of 300 uncomplicated cases of pleural effusion in the Massachusetts General Hospital, followed by R. C. Cabot, the subsequent history was ascertained in 221; followed five years until death or phthisis, 117; well after five years, 96.

In 172 of the cases of pleurisy with effusion in the Johns Hopkins Hospital Hamman got reports from 88; of these 48 were living and well, 30 later became tuberculous, in 2 the result was questionable, and 8 died of other diseases. Twelve of the 88 had tubercle bacilli in the sputum while in the hospital without discoverable pulmonary lesion; 3 of the 12 were living and well; in 8 the signs became well marked; one died of unknown cause. Hamman has collected 562 cases (including the above) in which the subsequent history was sought; of these 167, 29.7 per cent. became tuberculous.

Bacteriology of Acute Pleurisy.—From a bacteriological standpoint we may recognize three groups of cases, caused by the tubercle bacillus, the pneumococcus, and the streptococcus, respectively.

Bacillus tuberculosis is present in a very large proportion of all cases of primary or so-called idiopathic pleurisy. The exudate is usually sterile on cover slips or in the culture and inoculation tests made in the ordinary way, as the bacilli are very scanty. It has been demonstrated clearly that a large

amount of the exudate must be taken to make the test complete, either in cultures or in the inoculation of animals. Eichhorst found that more than 62 per cent. were demonstrated as tuberculous when as much as 15 c. c. of the exudate was inoculated into test animals, while less than 10 per cent. of the cases showed tuberculosis when only 1 c. c. of the exudate was used. This is a point to which observers should pay very special attention. Le Damany has demonstrated the tuberculous character of all but 4 in 55 primary pleurisies. He used large quantities of the fluid for his inoculation experiments.

The pneumococcus pleurisy is almost always secondary to a focus of inflammation in the lung. It may, however, be primary. The exudate is usually purulent and the outlook is very favorable.

The streptococcus pleurisy is the typical septic form which may occur either from direct infection of the pleura through the lung in broncho-pneumonia, or in cases of streptococcus pneumonia; in other instances it follows infection of more distant parts. The acute streptococcus pleurisy is the most serious and fatal of all forms.

Among other bacteria which have been found are the staphylococcus, Friedländer's bacillus, the typhoid bacillus, and the diphtheria bacillus.

Morbid Anatomy.—In sero-fibrinous pleurisy the serous exudate is abundant and the fibrin is found on the pleural surfaces and scattered through the fluid in the form of flocculi. The proportions of these constituents vary a great deal. In some instances there is very little membranous fibrin; in others it forms thick, creamy layers and exists in the dependent part of the fluid as whitish, curd-like masses. The fluid ~~of sero-fibrinous pleurisy~~ is of a lemon color, either clear or slightly turbid, depending on the number of formed elements. In some instances it has a dark brown color. The microscopic examination ~~of the fluid~~ shows leucocytes, occasional swollen cells, which may possibly be derived from the pleural endothelium, shreds of fibrillated fibrin, and a variable number of red blood-corpuscles. A large number of cells undergoing mitotic division is diagnostic of malignant disease. ~~On boiling,~~ the fluid is ~~found to be~~ rich in albumin. ~~Sometimes~~ it coagulates spontaneously. Its composition closely resembles that of blood serum. Cholesterin, uric acid, and sugar are occasionally found. The amount of the effusion varies from ½ to 4 litres. Enormous amounts are sometimes removed, 188 ounces in one case (E. C. Carter). The lung in ~~acute~~ sero-fibrinous pleurisy is more or less compressed. If the exudation is limited the lower lobe alone is atelectatic; but in an extensive effusion which reaches to the clavicle the entire lung will be found lying close to the spine, dark and airless, or even bloodless—*i. e.*, carnified.

In large exudations the adjacent organs are displaced; the liver is depressed and the heart dislocated. With reference to the position of the heart, the following statements may be made: (1) Even in the most extensive left sided exudation there is no rotation of the apex of the heart, which in no case was to the right of the mid-sternal line; (2) the relative position of the apex and base is usually maintained; in some instances the apex is lifted, in others the whole heart lies more transversely; (3) the right chambers of the heart occupy the greater portion of the front, so that the displacement is rather a definite dislocation of the mediastinum, with the pericardium, to the right, than any special twisting of the heart itself; (4) the kink or twist in

the inferior vena cava described by Bartels was not present in any of my cases.

Symptoms.—Prodromes are not uncommon, but the disease may set in abruptly with a chill, followed by fever and a severe pain in the side. In very many cases, however, the onset is insidious, particularly in children and in elderly persons. A little dyspnœa on exertion and an increasing pallor may be the only features. Washbourn has called attention to the frequency with which the pneumococcus pleurisy sets in with the features of pneumonia. The pain in the side is the most distressing symptom, and is usually referred to the nipple or axillary regions. It must be remembered, however, that pleuritic pain may be felt in the abdomen or low down in the back, particularly when the diaphragmatic surface of the pleura is involved. It is lancinating, sharp, and severe, and is aggravated by cough. At this early stage, on auscultation, sometimes indeed on palpation, a dry friction rub can be detected. The fever rarely rises so rapidly as in pneumonia, and does not reach the same grade. A temperature of from 102° to 103° F. is an average pyrexia. It may drop to normal at the end of a week or ten days without the appearance of any definite change in the physical signs, or it may persist for several weeks. The temperature of the affected is higher than that of the sound side. Cough is an early symptom in acute pleurisy, but is rarely so distressing or so frequent as in pneumonia. There are instances in which it is absent. The expectoration is usually slight in amount, mucoid, in character, and occasionally streaked with blood.

At the outset there may be dyspnœa, due partly to the fever and partly to the pain in the side. Later it results from the compression of the lung, particularly if the exudation has taken place rapidly. In the cases with very rapid effusion the dyspnœa may be very marked. When, however, the fluid is effused slowly, one lung may be entirely compressed without inducing shortness of breath, except on exertion, and the patient will lie quietly in bed without evincing the slightest respiratory distress. When the effusion is large the patient usually prefers to lie upon the affected side.

Physical Signs.—Inspection shows some degree of immobility on the affected side, depending upon the amount of exudation; and in large effusions an increase in volume, which may appear to be much more than it really is as determined by mensuration. The intercostal depressions are obliterated. In the right sided effusions the apex beat may be lifted to the fourth interspace or be pushed beyond the left nipple, or may even be seen in the axilla. When the exudation is on the left side, the heart's impulse may not be visible; but if the effusion is large it is seen in the third and fourth spaces on the right side, and sometimes as far out as the nipple, or even beyond it.

Palpation enables us more successfully to determine the deficient movements on the affected side, and the obliteration of the intercostal spaces, and more accurately to define the position of the heart's impulse. In simple serofibrinous effusion there is rarely any œdema of the chest walls. It is scarcely ever possible to obtain fluctuation. Tactile fremitus is greatly diminished or abolished. If the effusion is slight there may be only enfeeblement. The absence of the voice vibrations in effusions of any size constitutes one of the most valuable of physical signs. In children there may be much effusion with

retention of fremitus. In rare cases the vibrations may be communicated to the chest walls through localized pleural adhesions.

Mensuration.—With the cyrtometer, if the effusion is excessive, a difference of from half an inch to an inch, or even, in large effusions, an inch and a half, may be found between the two sides. Allowance must be made for the fact that the right side is naturally larger than the left. With the saddle tape the difference in expansion ~~between the two sides~~ can be conveniently measured.

Percussion.—Early in the disease there may be no alteration in the note, but with the gradual accumulation of the fluid the resonance becomes defective, and finally gives place to absolute flatness. From day to day the gradual increase in height of the fluid may be studied. In a pleuritic effusion rising to the fourth rib in front the percussion signs are usually very suggestive. In the subclavicular region the attention is often aroused at once by a tympanitic note, the so-called Skoda's resonance, which is heard perhaps more commonly in this situation with pleural effusion than in any other condition. It shades insensibly into a flat note in the lower mammary and axillary regions. Skoda's resonance may be obtained also behind, just above the limit of effusion. The dulness has a peculiarly resistant, wooden quality, differing from that of pneumonia and readily recognized by skilled fingers. It has long been known that when the patient is in the erect posture the upper line of dulness is not horizontal, but is higher behind than it is in front, forming a parabola. The curve marking the intersection of the plane of contact of lung and fluid with the chest wall is known as "Ellis's line of flatness," which Garland has verified clinically and by animal experiments. With medium-sized effusions this line begins lowest behind, advances upward and forward in a letter-S curve to the axillary region, whence it proceeds in a straight decline to the sternum. This curve is demonstrable only when the patient is in the erect position. Grocco, in 1902, called attention to the existence in pleural effusion of a triangular area of relative dulness, along the spine, on the side opposite to the pleurisy, in width from 2 to 5 cm., and with the apex upward. It can be demonstrated in a large majority of all cases, particularly in the young and in thin persons. It is possibly due to the bulging of the mediastinum, by the fluid, across the middle line, the anatomical possibility of which has been pointed out by Calvert.

On the right side the dulness passes without change into that of the liver. On the left side in the nipple line it extends to and may obliterate Traube's semilunar space. If the effusion is moderate, the phenomenon of movable dulness may be obtained by marking carefully, in the sitting posture, the upper limit in the mammary region, and then in the recumbent posture, noting the change in the height of dulness. This infallible sign of fluid can not always be obtained. In very copious exudation the dulness may reach the clavicle and even extend beyond the sternal margin of the opposite side.

Auscultation.—Early in the disease a friction rub ~~can usually~~ be heard, which disappears as the fluid accumulates. It is a to-and-fro dry rub, close to the ear, and has a leathery, creaking character. There is another pleural friction sound which closely resembles, and is scarcely to be distinguished from, the fine crackling crepitus of pneumonia. This may be heard at the commencement of the disease, and also, as pointed out in 1844 by MacDonnell,

May 1

Sr., of Montreal, when the effusion has receded and the pleural layers come together again.

With even a slight exudation there is weakened or distant breathing. Often inspiration and expiration are distinctly audible, though distant, and have a tubular quality. Sometimes only a puffing tubular expiration is heard, which may have a metallic or amphoric quality. Loud resonant râles accompanying this may forcibly suggest a cavity. These pseudo-cavernous signs are met with more frequently in children, and often lead to error in diagnosis. Above the line of dulness the breath sounds are usually harsh and exaggerated, and may have a tubular quality.

The vocal resonance is usually diminished or absent. The whispered voice is said to be transmitted through a serous and not through a purulent exudate (Baccelli's sign), but this is not always true. This author advises direct auscultation in the antero-lateral region of the chest. There may, however, be intensification—bronchophony. The voice sometimes has a curious nasal, squeaking character, which was termed by Laennec *ægophony*, from its supposed resemblance to the bleating of a goat. In typical form this is not common, but it is by no means rare to hear a curious twang-like quality in the voice, particularly at the outer angle of the scapula.

In the examination of the heart ~~in cases of pleuritic effusion~~ it is well to bear in mind that when the apex of the heart lies beneath the sternum there may be no impulse. The determination of the situation of the organ may rest with the position of maximum loudness of the sounds. Over the displaced organ a systolic murmur may be heard. When the lappet of lung over the pericardium is involved on either side there may be a pleuro-pericardial friction.

Blood Count in Pleural Effusion.—Emerson ~~has~~ looked over ~~for me~~ the histories of 89 cases of acute pleurisy with effusion in which the blood counts were made before the temperature reached normal. Only 26 had a leucocytosis between 10,000 and 15,000; one only above 15,000. In 12 of the cases the count was below 5,000.

The X-ray pictures are of great interest and of much value in diagnosis. They ~~have~~ shown that the effusion is not always in the lower portion of the chest with the patient in the upright position, but that it may represent a vertical column in the lateral aspect of the chest, compressing the lung toward the spine. The effusion is not always mobile, but may be fixed by adhesions in one position.

Course.—The course of acute sero-fibrinous pleurisy is very variable. After persisting for a week or ten days the fever subsides, the cough and pain disappear, and a slight effusion may be quickly absorbed. In cases in which the effusion reaches as high as the fourth rib recovery is usually slower. Many instances come under observation for the first time, after two or three weeks' indisposition, with the fluid at a level with the clavicle. The fever may last from ten to twenty days without exciting anxiety, though, as a rule, in ordinary pleurisy from cold, as we say, the temperature in cases of moderate severity is normal within eight or ten days. Left to itself, the natural tendency is to resorption; but this may take place very slowly. With the absorption of the fluid there is a redux-friction crepitus, either leathery and creaking or crackling and râle-like, and for months, or even longer, the

defective resonance and feeble breathing are heard at the base. Rare modes of termination are perforation and discharge through the lung, and externally through the chest wall, examples of which have been recorded by Sahli.

The immediate prognosis in pleurisy with effusion is good. Of 320 cases at St. Bartholomew's Hospital, only 6.1 per cent. died before leaving the hospital (Hedges).

A sero-fibrinous exudate may persist for months without change, particularly in tuberculous cases, and will sometimes reaccumulate after aspiration and resist all treatment. After persistence for more than twelve months, in spite of repeated tapping, a serous effusion was cured by incision without deformity of the chest (S. West). When one pleura is full and the heart is greatly dislocated, the condition, although in a majority of cases producing remarkably little disturbance, is not without risk.

3. PURULENT PLEURISY

(*Empyema*)

Etiology.—Pus in the pleura is due to (*a*) infection from within, as a rule directly from a patch of pneumonia or a septic focus due to the pneumococcus or the pus organisms, in some cases a tuberculous broncho-pneumonia; (*b*) involvement from without, as in fracture of a rib, penetrating wound, disease of œsophagus, etc.

It frequently follows the infectious diseases, particularly scarlet fever. It is very often latent, and due to undiscovered foci of lobar or lobular pneumonia. It is common in children, more in boys than in girls, and between the ages of one and five and eight and nine.

The pneumococcus is the most common organism, then the ordinary pus organisms and the tubercle bacilli. The pneumococcus has been found and in rare cases the influenza bacillus, and even psorosperms.

Morbid Anatomy.—On opening an empyema post mortem we usually find that the effusion has separated into a clear, greenish yellow serum above and the thick, cream-like pus below. The fluid may be scarcely more than turbid, with flocculi of fibrin through it. In the pneumococcus empyema the pus is usually thick and creamy. It usually has a heavy, sweetish odor, but in some instances—particularly those following wounds—it is fetid. In cases of gangrene of the lung or pleura the pus has a horribly stinking odor. Microscopically it has the characters of ordinary pus. The pleural membranes are greatly thickened, and present a grayish white layer from 1 to 2 mm. in thickness. On the costal pleura there may be erosions, and in old cases fistulous communications are common. The lung may be compressed to a very small limit, and the visceral pleura also may show perforations.

Symptoms.—Purulent pleurisy may begin abruptly, with the symptoms already described. More frequently it comes on insidiously in the course of other diseases or follows an ordinary sero-fibrinous pleurisy. There may be no pain in the chest, very little cough, and no dyspnœa, unless the side is very full. Symptoms of septic infection are rarely wanting. If in a child, there is a gradually developing pallor and weakness; sweats occur, and there is irregular fever. A cough is by no means constant. The leucocytes are

usually much increased; in one fatal case they numbered 115,000 per ~~cubic millimetre~~ cmm.

Physical Signs.—Practically they are those already considered in pleurisy with effusion. There are, however, one or two additional points to be mentioned. In empyema, particularly in children, the disproportion between the sides may be extreme. The intercostal spaces may not only be obliterated, but may bulge. Not infrequently there is œdema of the chest walls. The network of subcutaneous veins may be very distinct. It must not be forgotten that in children the breath-sounds may be *loud and tubular* over a purulent effusion of considerable size. The dislocation of the heart and the displacement of the liver are more marked in empyema than in sero~~fibrinous~~ effusion—probably, as Senator suggests, owing to the greater weight of the fluid.

A curious phenomenon associated generally with empyema, but sometimes occurring in the sero-fibrinous exudate, is *pulsating pleurisy*, first described by MacDonnell, Sr., of Montreal. In 95 cases collected by Sailer it was much more frequent in males than in females. In 38 there was a tumor; that is, empyema necessitatis. In all but one case the fluid was purulent. Pneumothorax may be present. There are two groups of cases, the intrapleural pulsating pleurisy and the pulsating *empyema necessitatis*, in which there is an external pulsating tumor. No satisfactory explanation has been offered how the heart impulse is thus forcibly communicated through the effusion.

Empyema is a chronic affection, which in a few instances terminates naturally in recovery, but a majority of cases, if left alone, ends in death. The following are some modes of natural cure: (*a*) By absorption of the fluid. In small effusions this may take place gradually. The chest wall sinks. The pleural layers become greatly thickened and enclose between them the inspissated pus, in which lime salts are gradually deposited. Such a condition may be seen once or twice a year in the post mortem room of any large hospital. (*b*) By perforation of the lung. Although in this event death may take place rapidly, by suffocation, as Aretæus says, yet in cases in which it occurs gradually recovery may follow. ~~Since 1873, when I saw a case of this kind in Traube's clinic, and heard his remarks on the subject, I~~ have seen a number of instances of the kind and can corroborate ~~his~~ statement as to the favorable termination of many of them. Empyema may discharge either by opening into the bronchus and forming a fistula, or, as Traube pointed out, by producing necrosis of the pulmonary pleura, sufficient to allow the soaking of the pus through the spongy lung tissue into the bronchi. In the first way pneumothorax usually, though not always, develops. In the second way the pus is discharged, without formation of pneumothorax. Even with a bronchial fistula recovery is possible. (*c*) By perforation of the chest wall—*empyema necessitatis*. This is by no means an unfavorable method, as many cases recover. The perforation may occur anywhere in the chest wall, but is, as Cruveilhier remarked, more common in front. It may be anywhere from the third to the sixth interspace, usually, according to Marshall, in the fifth. It may perforate in more than one place, and there may be a fistulous communication which opens into the pleura at some distance from the external orifice. The tumor, when near the heart, may pulsate. The discharge may persist for years. In Copeland's Dictionary is mentioned an instance of a

Bavarian physician who had a pleural fistula for thirteen years and enjoyed fairly good health.

An empyema may perforate the neighboring organs, the œsophagus, peritoneum, pericardium, or the stomach. A remarkable sequel is a pleuro-œsophageal fistula, of which cases have been reported by Voelcker, Thursfield, and myself. In my case there was a fistulous communication through the chest wall. Very remarkable cases are those which pass down the spine and along the psoas into the iliac fossa, and simulate a psoas or lumbar abscess.

4. TUBERCULOUS PLEURISY

This has already been considered (p. 178), and the symptoms and physical signs do not require any description other than that already given in connection with the sero-fibrinous and purulent forms.

5. OTHER VARIETIES OF PLEURISY

Hæmorrhagic Pleurisy.—A bloody effusion is met with under the following conditions: (*a*) In the pleurisy of asthenic states, such as cancer, Bright's disease, and occasionally in the malignant fevers. It is interesting to note the frequency with which hæmorrhagic pleurisy is found in cirrhosis of the liver. It occurred in the very patient in whom Laennec first accurately described this disease. While this may be a simple hæmorrhagic pleurisy, in a majority of the cases which I have seen it has been tuberculous. (*b*) Tuberculous pleurisy, in which the bloody effusion may result from the rupture of newly formed vessels in the soft exudate accompanying the eruption of miliary tubercles, or it may come from more slowly formed tubercles in a pleurisy secondary to extensive pulmonary disease. (*c*) Cancerous pleurisy, whether primary or secondary, is frequently hæmorrhagic. (*d*) Occasionally hæmorrhagic exudation is met with in perfectly healthy individuals, in whom there is not the slightest suspicion of tuberculosis or cancer. In one such case, a large, able-bodied man, the patient was to my knowledge healthy and strong eight years afterward. And, lastly, it must be remembered that during aspiration the lung may be wounded and blood in this way get mixed with the sero-fibrinous exudate. The condition of hæmorrhagic pleurisy is to be distinguished from hæmothorax, due to the rupture of aneurism or the pressure of a tumor on the thoracic veins.

Diaphragmatic Pleurisy.—The inflammation may be limited partly or chiefly to the diaphragmatic surface. This is often a dry pleurisy, but there may be effusion, either sero-fibrinous or purulent, which is circumscribed on the diaphragmatic surface. In these cases the pain is low in the zone of the diaphragm and may simulate that of acute abdominal disease. It may be intensified by pressure at the point of insertion of the diaphragm at the tenth rib. The diaphragm is fixed and the respiration is thoracic and short. Andral noted in certain cases severe dyspnœa and attacks simulating angina. As mentioned, the effusion is usually plastic, not serous. Serous or purulent effusions of any size limited to the diaphragmatic surface are extremely rare. Intense subjective with trifling objective features are always suggestive of diaphragmatic pleurisy.

Encysted Pleurisy.—The effusion may be circumscribed by adhesions or separated into two or more pockets or loculi, which communicate with each other. This is most common in empyema. In these cases there have usually been, at different parts of the pleura, multiple adhesions by which the fluid is limited. In other instances the recent false membranes may encapsulate the exudation on the diaphragmatic surface, for example, or the part of the pleura posterior to the mid-axillary line. The condition may be very puzzling during life, and present special difficulties in diagnosis. In some cases the tactile fremitus is retained along certain lines of adhesion. The exploratory needle should be freely used.

Interlobar pleurisy forms an interesting and not uncommon variety. In nearly every instance of acute pleurisy the interlobar serous surfaces are also involved and closely agglutinated together, and sometimes the fluid is encysted between them. In this position tubercles are to be carefully looked for. In a case of this kind following pneumonia there was between the lower and upper and middle lobes of the right side an enormous purulent collection, which looked at first like a large abscess of the lung. These collections may perforate the bronchi, and the cases present special difficulties in diagnosis.

Diagnosis of Pleurisy

Acute plastic pleurisy is readily recognized. In the diagnosis of pleuritic effusion the first question is, Does a fluid exudate exist? the second, What is its nature? In large effusions the increase in the size of the affected side, the immobility, the absence of tactile fremitus, together with the displacement of organs, give infallible indications of the presence of fluid. The chief difficulty arises in effusions of moderate extent, when the dulness, the presence of bronchophony, and, perhaps, tubular breathing may simulate *pneumonia.* The chief points to be borne in mind are: (*a*) Differences in the onset and in the general characters of the two affections, more particularly the initial chill, the higher fever, more urgent dyspnœa, and the rusty expectoration, which characterize pneumonia. As already mentioned, some of the cases of pneumococcus pleurisy set in like pneumonia. (*b*) Certain physical signs—the more wooden character of the dulness, the greater resistance, and the marked diminution or the absence of tactile fremitus in pleurisy. The auscultatory signs may be deceptive. It is usually, indeed, the persistence of tubular breathing, particularly the high-pitched, even amphoric expiration, heard in some cases of pleurisy, which has raised the doubt. The intercostal spaces are more commonly obliterated in pleuritic effusion than in pneumonia. ~~As already mentioned,~~ the displacement of organs is a very valuable sign. Nowadays with the ~~hypodermic~~ needle the question is easily settled. ~~A separate small syringe with a capacity of two drachms should be reserved for exploratory purposes, and the needle should be longer and firmer than in the ordinary hypodermic instrument.~~ With careful preliminary disinfection the instrument can be used with impunity, and in cases of doubt the exploratory puncture should be made without hesitation. Pneumothorax is an occasional sequence. The ~~hypodermic~~ needle is especially useful in those cases in which there are pseudo-cavernous signs at the base. In cases, too, of massive pneumonia, in which the bronchi are plugged with fibrin, if the patient has not been seen from the outset, the diagnosis may be impossible without it.

On the left side it may be difficult to differentiate a very large pericardial from a pleural effusion. The retention of resonance at the base, the presence of Skoda's resonance toward the axilla, the absence of dislocation of the heart-beat to the right of the sternum, the feebleness of the pulse and of the heart-sounds, and the urgency of the dyspnœa, out of all proportion to the extent of the effusion, are the chief points to be considered. Unilateral hydrothorax, which is not at all uncommon in heart-disease, presents signs identical with those of sero-fibrinous effusion. Certain tumors within the chest may simulate pleural effusion. It should be remembered that many intrathoracic growths are accompanied by exudation. Malignant disease of the lung and of the pleura and hydatids of the pleura produce extensive dulness, with suppression of the breath-sounds, simulating closely effusion.

On the right side, abscess of the liver, subdiaphragmatic abscess, and hydatid cysts may rise high into the pleura and produce dulness and enfeebled breathing. Often in these cases there is a friction sound, which should excite suspicion, and the upper outline of the dulness is sometimes plainly convex. In a case of cancer of the kidney the growth involved the diaphragm very early, and for months there were signs of pleurisy before our attention was directed to the kidney. In all these instances the exploratory puncture should be made.

The second question, as to the nature of the fluid, is quickly decided by the use of the needle. The persistent fever, the occurrence of sweats, a leucocytosis, and the increase in the pallor suggest the presence of pus. In children the complexion is often sallow and earthy. In protracted cases, even in children, when the general symptoms and the appearance of the patient have been most strongly suggestive of pus, the syringe has withdrawn clear fluid. On the other hand, effusions of short duration may be purulent, even when the general symptoms do not suggest it. In pneumonia the practitioner should be on the alert if the crisis is delayed or the temperature rises after the crisis, if chills and sweats follow, or if the cough changes to one of paroxysmal type of great intensity. There are three groups: (*a*) The presence of the empyema is readily detected. (*b*) It is suspected, but it is not possible to locate the pus by the ordinary physical means. The exploratory needle should be freely used with the aid of a local anæsthetic; ~~many punctures may be made without discomfort~~. (*c*) In a few instances small interlobar collections, small mural abscesses, and the diaphragmatic form may escape detection until an operation is performed. The prognostic import of the bacteriological examination of the aspirated fluid is as follows: The pneumococcus is of favorable significance, as such cases usually get well rapidly, even with a single aspiration. The streptococcus empyema is the most serious form, and even after a free drainage the patient may succumb to a general septicæmia. A sterile fluid indicates in a majority of instances a tuberculous origin.

Treatment

At the onset the severe pain may be relieved by hot or cold applications, but a hypodermic of morphia is more effective. The Paquelin cautery may be lightly but freely applied. It is well to administer a mercurial or saline purge. Fixing the side by careful strapping with long strips of adhesive plaster, which

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should pass well over the middle line, drawn tightly and evenly, gives great relief, ~~and I can corroborate the statement of F. T. Roberts as to its efficacy~~. Cupping, wet or dry, is now seldom employed. Blisters are of no special service in the acute stages, although they relieve the pain. The ice-bag may be used as in pneumonia. The open-air treatment should be begun early, as a majority of the cases are tuberculous. Medicines are rarely required and mercurials are not indicated. A Dover's powder may be given at night.

When effusion has taken place, mustard plasters or iodine, producing slight counter-irritation, appear useful, particularly in the later stages. Iodide of potassium is of doubtful benefit. By some the salicylates are believed to be of special efficacy; but the drug treatment of the disease is most unsatisfactory. The dry diet and frequent saline purges (given in concentrated form before breakfast ~~in Hay's method~~) may be tried. ~~Recently~~ It has been advised to use a salt-free diet.

Early and if necessary repeated aspiration of the fluid is the most satisfactory ~~method of~~ treatment. The results obtained by Delafield in 200 cases treated by early aspiration have never been equalled by any other method. The credit of introducing aspiration in pleuritic effusions is due to Morrill Wyman, of Cambridge, Mass., and Henry I. Bowditch, of Boston. Years prior to Dieulafoy's work, aspiration was in constant use at the Massachusetts General Hospital and was advocated repeatedly by Bowditch. As the question is one of some historical interest, I give Bowditch's conclusions concerning aspiration, expressed more than sixty years ago, and which practically represent the opinion of to-day: "(1) The operation is perfectly simple, but slightly painful, and can be done with ease upon any patient in however advanced a stage of the disease. (2) It should be performed forthwith in *all* cases in which there is complete filling up of one side of the chest. (3) He had determined to use it in *any* case of even *moderate* effusion lasting more than a few weeks and in which there should seem to be a disposition to resist ordinary modes of treatment. (4) He urged this practice upon the profession as a very important measure in practical medicine; believing that by this method death may frequently be prevented from ensuing either by sudden attack of dyspnœa or subsequent phthisis, and, finally, from the gradual wearing out of the powers of life or inability to absorb the fluid." When the fluid reaches to the clavicle the indication for aspiration is imperative. Fever is not a contra-indication; indeed, sometimes with serous exudates the temperature falls after aspiration.

The operation is extremely simple and is practically without risk. The spot selected for puncture should be either in the sixth intercostal space in the mid-axilla or at the outer angle of the scapula in the eighth space. The arm of the patient should be brought forward with the hand on the opposite shoulder, so as to widen the spaces. The needle should be thrust in close to the upper margin of the rib, so as to avoid the intercostal artery, the wounding of which, however, is an exceedingly rare accident. The fluid should be withdrawn slowly. The amount will depend on the size of the exudate. If the fluid reaches to the clavicle a litre or more may be withdrawn with safety. In chronic cases of serous pleurisy after the failure of repeated tappings S. West has shown the great value of free incision and drainage. He has reported cases of recovery after effusions of fifteen and eighteen months' standing.

Repeated tapping may be required in some cases. In the chronic cases the injection of adrenalin solution (20 to 30 drops of a 1 to 1,000 solution) into the pleural cavity after aspiration has proved of value.

Symptoms and Accidents during Paracentesis.—Pain is usually complained of after a certain amount of fluid has been withdrawn; it is sharp and cutting in character. *Coughing* occurs toward the close, and may be severe and paroxysmal. *Pneumothorax* may follow an exploratory puncture with a hypodermic needle; it is rare during aspiration. *Subcutaneous emphysema* may develop from the point of puncture, without the production of pneumothorax. *Cerebral symptoms.*—Faintness is not uncommon. Epileptic convulsions may occur either during the withdrawal or while irrigating the pleura. These symptoms are very difficult to explain and are regarded by most authors as of reflex origin. Hemiplegia may follow. And lastly *sudden death* may occur either from syncope or during the convulsions.

As A. E. Russell has pointed out, these serious and even fatal events may follow exploratory puncture of the lung. Such accidents of paracentesis and of washing out the pleura are explained by the studies of Capps and Lewis, who have shown that a sudden and sometimes fatal fall in blood pressure may follow the experimental irrigation of the pleura in dogs. Occasionally toxic symptoms arise resembling those of the "serum illness"—pains in the joints, albumin in the urine, and œdema—suggestive of the absorption of toxins that act like a heterogenous serum. Expectoration of a large quantity of *albuminous fluid* may occur suddenly after the tapping, associated with dyspnœa. Some cases have proved rapidly fatal, with the features of an acute œdema of the lungs. It has occurred only once in my practice.

The after-treatment of pleurisy is important and the patients should be handled exactly as if they had an early tuberculous lung lesion.

Empyema.—A majority of the cases get well, provided that free drainage is obtained, and it makes no difference practically what measures are followed so long as this indication is met. The good results in any method depend upon the thoroughness with which the cavity is drained. Irrigation of the cavity is rarely necessary unless the contents are fetid. In the subsequent treatment a point of great importance in facilitating the closure of the cavity is the distention of the lung on the affected side. This may be accomplished by the method advised by Ralston James, which has been practiced with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily, for a certain length of time, increasing gradually with the increase of his strength, transfers by air-pressure water from one bottle to another. The bottles should be large, holding at least a gallon each, and by the arrangement of tubes, as in the Wolff's bottle, an expiratory effort of the patient forces the water from one bottle into the other. Equally efficacious is the plan advised by Naunyn. The patient sits in an arm-chair grasping strongly one of the rungs with the hand and forcibly compressing the sound side against the arm of the chair; then forcible inspiratory efforts are made which act chiefly on the compressed lung, as the sound side is fixed. The abscess cavity is gradually closed, partly by the falling in of the chest wall and partly by the expansion of the lung. In some instances it is necessary to resect portions of one or more ribs.

Until recently efficient drainage has been regarded as the most important

Cases of effusion but serofibrinous & purulent.

if treated intensively only an excess of Poly. Leucocytes may clear by aspiration. (15th 27 at N.S.H. Ward)

relatively favourable if pneumococci.

pur. less " " streptococci.

consideration, and both operative and drainage proceedings have been directed toward making the chest wall conform to the lung. While thoracotomy and free drainage have done a great deal, it must be confessed that in a not inconsiderable number of cases the obliteration of the pus cavity has been a long and sometimes hopeless matter. In its place continuous drainage and intermittent siphonage have been used. It looks as if surgeons have made an important departure in the method of negative tension drainage as devised by von Eberts.

The physician is often asked, in cases of empyema with emaciation, ~~hectic~~ fever and feeble, rapid pulse, whether the patient could stand the operation. Even in the most desperate cases the surgeon should never hesitate to make a free incision.

II. CHRONIC PLEURISY

This affection occurs in two forms:

Chronic pleurisy with effusion in which the disease may set in insidiously or may follow an acute sero-fibrinous pleurisy. There are cases in which the ~~liquid~~ persists for months or even years without undergoing any special alteration and without becoming purulent. Such cases have the characters which we have described under pleurisy with effusion.

Chronic Dry Pleurisy.—The cases are met with (*a*) *as a sequence of ordinary pleural effusion.* When the exudate is absorbed and the layers of the pleura come together there is left between them a variable amount of fibrinous material which gradually undergoes organization, and is converted into a layer of firm connective tissue. This process goes on at the base, and is represented clinically by a slight grade of flattening, deficient expansion, defective resonance on percussion, and enfeebled breathing. After recovery from empyema the flattening and retraction may be still more marked. In both cases it is a condition which can be greatly benefited by pulmonary gymnastics. In these firm, fibrous membranes calcification may occur, particularly after empyema. It is not very uncommon to find between the false membranes a small pocket of fluid forming a sort of pleural cyst. In the great majority of these cases the condition is one which need not cause anxiety. There may be an occasional dragging pain at the base of the lung or a stitch in the side, but patients may remain in perfectly good health for years. The most advanced grade of this secondary dry pleurisy is seen in those cases of empyema which have been left to themselves and have perforated and ultimately healed by a gradual absorption or discharge of the pus, with retraction of the side of the chest and permanent carnification of the lung. Traumatic lesions, such as gunshot wounds, may be followed by an identical condition. Post mortem, it is quite impossible to separate the layers of the pleura, which are greatly thickened, particularly at the base, and surround a compressed, airless, fibroid lung. Bronchiectasis may gradually ensue, and in one remarkable case ~~which I saw~~ on several occasions with Blackader, of Montreal, not only on the affected side, but also in the lower lobe of the other lung.

(*b*) *Primitive dry pleurisy.*—This condition may directly follow the acute plastic pleurisy already described; but it may set in without any acute symptoms whatever, and the patient's attention may be called to it by feeling the

pleural friction. A constant effect of this primitive dry pleurisy is the adhesion of the layers. This is probably an invariable result, whether the pleurisy is primary or secondary. The organization of the thin layer of exudation in a pneumonia will unite the two surfaces by delicate bands. Pleural adhesions are extremely common, and it is rare to examine a body entirely free from them. They may be limited in extent or universal. Thin fibrous adhesions do not produce any alteration in the percussion characters, and, if limited, there is no special change heard on auscultation. When, however, there is general synechia on both sides the expansile movement of the lung is considerably impaired. We should naturally think that universal adhesions would interfere materially with the function of the lungs, but practically we see many instances in which there has not been the slightest disturbance. The physical signs of total adhesion are by no means constant. It has been stated that there is a marked disproportion between the degree of expansion of the chest walls and the intensity of the vesicular murmur, but the latter is a very variable factor, and under perfectly normal conditions the breath-sounds, with very full chest expansion, may be extremely feeble. The diaphragm phenomenon—Litten's sign—is absent.

As already stated, it is possible, as the late Sir Andrew Clark held, that a primitive dry pleurisy may gradually lead to great thickening of the membranes, and ultimate invasion of the lung, causing a cirrhosis.

Lastly, there is a primitive dry pleurisy of tuberculous origin. In it both parietal and costal layers are greatly thickened—perhaps from 2 to 3 mm. each—and present firm fibroid, caseous masses and small tubercles, while uniting these two greatly thickened layers is a reddish-gray fibroid tissue, sometimes infiltrated with serum. This may be a local process confined to one pleura, or it may be in both. These cases are sometimes associated with a similar condition in the pericardium and peritoneum.

Occasionally remarkable vaso-motor phenomena occur in chronic pleurisy, whether simple or in connection with tuberculosis of an apex. Flushing or sweating of one cheek or dilatation of the pupil are the common manifestations. They appear to be due to involvement of the first thoracic ganglion at the top of the pleural cavity.

III. HYDROTHORAX

Hydrothorax is a transudation of simple non-inflammatory fluid into the pleural cavities, and occurs as a secondary process in many affections. The fluid is clear, without any flocculi of fibrin, and the membranes are smooth. It is met with more particularly in connection with general dropsy, either renal, cardiac, or hæmic. It may, however, occur alone, or with only slight œdema of the feet. A child was admitted to the Montreal General Hospital with urgent dyspnœa and cyanosis, and died the night after admission. She had extensive bilateral hydrothorax, which had come on early in the nephritis of scarlet fever. In renal disease hydrothorax is almost always bilateral, but in heart affections one pleura is more commonly involved. The physical signs are those of pleural effusion, but the exudation is rarely excessive. In kidney and heart-disease, even when there is no general dropsy, the occurrence of dyspnœa should at once direct attention to the pleura, since many patients

are carried off by a rapid effusion. In chronic valvular disease the effusion is usually on the right side, and may recur for months. Stengel attributes the greater frequency of the dextral effusion to compression of the azygos veins. Post mortem records show the frequency with which this condition is overlooked. The saline purges will in many cases rapidly reduce the effusion, but, if necessary, aspiration should be practiced repeatedly.

IV. PNEUMOTHORAX

(*Hydro-Pneumothorax and Pyo-Pneumothorax*)

Air alone in the pleural cavity, to which the term pneumothorax is strictly applicable, is an extremely rare condition. It is almost invariably associated with a serous fluid—hydro-pneumothorax, or with pus—pyo-pneumothorax.

Etiology.—There exists normally within the pleural cavity of an adult a negative pressure of several (3 to 5) millimetres of mercury, due to the recoil of the distended, perfectly elastic lung. Hence, through any opening connecting the pleural cavity with the external air we should expect air to rush in until this negative pressure is relieved. To explain the absence of pneumothorax in a few cases of external injury laying the pleura bare, in which it would be expected, S. West has assumed the existence of a cohesion between the pleuræ, but this force has not as yet been satisfactorily demonstrated.

In a case of pneumothorax, if the opening causing it remain patent, which occurs only in some external wounds, or especially perforations through consolidated areas of the lungs, the intrathoracic pressure will be that of the atmosphere, the lung will be found to have collapsed as much as possible by virtue of its own elastic tension, the intercostal grooves obliterated, the heart displaced to the other side, and the diaphragm lower than normal, because the negative pressure by reason of which these organs are partly retained in their ordinary position has been relieved. If the opening becomes closed the intrathoracic pressure may rise above the atmospheric and the above-mentioned displacements be much increased. But most perforations through the lung are valvular, a property of lung tissue, and the intrapleural pressure is soon about 7 mm. of mercury. If there be a fluid exudate the pressure may be higher, but the high pressures supposed are more apparent than real, and that measured at the autopsy table is quite surely not that during life. It is more a question of the amount of distention than the actual pressure which determines the discomfort of the patient.

Pneumothorax arises: (1) In perforating wounds of the chest, in which case it is sometimes associated with extensive cutaneous emphysema. It may follow exploratory puncture either with a small needle or an aspirator. There were ten cases in my series. Pneumothorax rarely follows fracture of the rib, even though the lung may be torn. (2) In perforation of the pleura through the diaphragm, usually by malignant disease of the stomach or colon, or abscess of the liver perforating lung and pleura. The pleura may also be perforated in cases of cancer of the œsophagus. (3) When the lung is perforated. This is by far the most common cause, and may occur: (*a*) In the normal lung from rupture of the air-vesicles during straining or even when

at rest. Special attention has been called to this accident by S. West and De H. Hall. The air may be absorbed and no ill effect follow. It does not necessarily excite pleurisy, as pointed out many years ago by Gairdner, but inflammation and effusion are the usual result. In one of my cases the condition developed as the patient was going down-stairs; no effusion followed; he did not react to tuberculin. (*b*) From perforation due to local disease of the lung, either the softening of a caseous focus or the breaking of a tuberculous cavity. According to S. West, 90 per cent. of all the cases are due to this cause. Less common are the cases due to septic broncho-pneumonia and to gangrene. A rare cause is the breaking of a hæmorrhagic infarct in chronic heart-disease, of which I met an instance a few years ago. (*c*) Perforation of the lung from the pleura, which arises in certain cases of empyema and produces a pleuro-bronchial fistula. (4) Spontaneously, by the development in pleural exudates of the gas bacillus (*B. aërogenes capsulatus* Welch). Of 48 cases, the basis of Emerson's exhaustive monograph (J. H. H. Reports, vol. xi), 22 were tuberculous, 6 were the result of trauma, 10 of aspiration, 2 were spontaneous, 2 followed bronchiectasis, 2 abscess of the lung, 1 gangrene, 2 an empyema, and 1 abscess of the liver perforating through the lung.

Pneumothorax occurs chiefly in adults, though cases are met with in very young children. It is more frequent in males than in females.

A remarkable recurrent variety has been described by S. West, Goodhart, and Furney. In Goodhart's case the pneumothorax developed first in one side and then in the other.

Morbid Anatomy.—If the trocar or blow-pipe is inserted between the ribs, there may be a jet of air of sufficient strength to blow out a lighted match. On opening the thorax the mediastinum and pericardium are seen to be pushed, or rather, as Douglas Powell pointed out, "drawn over" to the opposite side; but, as before mentioned, the heart is not rotated, and the relation of its parts is maintained much as in the normal condition. A serous or purulent fluid is usually present, and the membranes are inflamed. The cause of the pneumothorax can usually be found without difficulty. In the great majority of instances it is the perforation of a tuberculous cavity or a breaking of a superficial caseous focus. The orifice of rupture may be extremely small. In chronic cases there may be a fistula of considerable size communicating with the bronchi. The lung is usually compressed and carnified.

Symptoms.—The onset is usually sudden and characterized by severe pain in the side, urgent dyspnœa, and signs of general distress, as indicated by slight lividity and a very rapid and feeble pulse—the pneumothorax acutissimus of Unverricht. There may, however, be no urgent symptoms, particularly in cases of long-standing phthisis.

Physical Signs.—The physical signs are very distinctive. *Inspection* shows marked enlargement of the affected side with immobility. The heart impulse is usually much displaced. On *palpation* the fremitus is greatly diminished or more commonly abolished. On *percussion* the resonance may be tympanitic or even have an amphoric quality. This, however, is not always the case. It may be a flat tympany, resembling Skoda's resonance. In some instances it may be a full, hyperresonant note, like emphysema; while in others—and this is very deceptive—there is dulness. These extreme variations depend doubtless upon the degree of intrapleural tension. On several occa-

sions I have known an error in diagnosis to result from ignorance of the fact that, in certain instances, the percussion note may be "muffled, toneless, almost dull" (Walshe). There is usually dulness at the base from effused fluid, which can readily be made to change the level by altering the position of the patient. Movable dulness can be obtained much more readily in pneumothorax than in a simple pleurisy. On *auscultation* the breath-sounds are suppressed. Sometimes there is only a distant feeble inspiratory murmur of marked amphoric quality. The contrast between the loud exaggerated breath-sounds on the normal side and the absence of the breath-sounds on the other is very suggestive. The râles have a peculiar metallic quality, and on coughing or deep inspiration there may be what Laennec termed the metallic tinkling. The voice, too, has a curious metallic echo. What is sometimes called the coin-sound, termed by Trousseau the *bruit d'airain,* is very characteristic. To obtain it the auscultator should place one ear on the back of the chest wall while the assistant taps one coin on another on the front of the chest. The metallic echoing sound which is produced in this way is one of the most constant and characteristic signs of pneumothorax. And, lastly, the Hippocratic succussion splash may be obtained when the auscultator's head is placed upon the chest while the patient's body is shaken. A splashing sound is produced, which may be audible at a distance. A patient may himself notice it in making abrupt changes in posture. The signs, distention, immobility, lack of vocal fremitus, hyperresonance, absence of breath-sounds and coin-sound, are those of the pure pneumothorax of Laennec. The metallic phenomena may be present, *e. g.,* the metallic tinkling and amphoric respiration, but these are best heard in cases with a consolidated lung and thickened pleura, such as occur in tuberculosis. The movable dulness and splash on succussion depend on fluid. Of other physical signs displacement of organs is most constant. As already mentioned, the heart may be much "drawn over" to the opposite side, and the liver greatly displaced, so that its upper surface is below the level of the costal margin, a degree of dislocation never seen in simple effusion.

Diagnosis.—The diagnosis of pneumothorax rarely offers any difficulty, as the signs are very characteristic. In cases in which the percussion note is dull the condition may be mistaken for effusion. Diaphragmatic or congenital hernia following a crush or other accident may closely simulate pneumothorax.

In cases of very large phthisical cavities with tympanitic percussion resonance and râles of an amphoric, metallic quality, the question of pneumothorax is sometimes raised. In those rare instances of total excavation of one lung the amphoric and metallic phenomena may be most intense, but the absence of dislocation of the organs, of the succussion splash, and of the coin-sound suffices to differentiate this condition. While this is true in the great majority of cases, I have heard the *bruit d'airain* over a large cavity in the right upper lobe. The condition of pyo-pneumothorax subphrenicus may simulate closely true pneumothorax.

The X-ray examination is of great help, and the picture in ordinary pneumothorax is very characteristic. There is a remarkable condition, described by Newton Pitt, associated with aneurism of the aorta, in which the sac pressing on the bronchus causes inflation of one lung with a picture simulating pneumothorax very closely.

Prognosis.—The prognosis in cases of pneumothorax depends largely upon the cause. S. West gives a mortality of 70 per cent. The tuberculous cases usually die within a few weeks. Of 39 cases, 29 died within a fortnight (West); 10 patients died on the first day, 2 within twenty and thirty minutes respectively of the attack. Of our 22 tuberculous cases 20 died, and 5 of the 10 cases following aspiration. Pneumothorax in a healthy individual often ends in recovery. There are tuberculous cases in which the pneumothorax, if occurring early, seems to arrest the progress of the tuberculosis. There is a chronic pneumothorax which may last for between three and four years. It may be a chronic condition, as in the case just mentioned, and a fair measure of health may be enjoyed.

Treatment.—There are three groups of cases: First, in the pneumothorax acutissimus, with urgent dyspnœa, great displacement of the heart, cyanosis, and low blood pressure, an opening should be made in the pleura and kept open, converting a valvular into an open variety. Immediate aspiration with a trocar has saved life. Secondly, the spontaneous cases which usually do well, as the air is quickly absorbed; so also with the traumatic variety. Very many of the tuberculous cases are best let alone, if the patient is doing well, or if the disease in the other lung is advanced. Thirdly, when there is pus, and the patient is not doing well, or in the tuberculous variety if the other lung is not involved, pleurotomy, or resection of one or two ribs, may be done. Of nine cases in ~~my~~ series two recovered.

V. AFFECTIONS OF THE MEDIASTINUM

Lymphadenitis.—The greater number of glands are on the right side, and the right bronchus passes off at a higher level (fifth dorsal vertebra) than the left. The glands are constantly enlarged in all inflammatory affections of the lungs. In all the acute affections of childhood they are found swollen. They are almost constantly involved in tuberculosis of the lungs and they are not infrequently the only ~~organs~~ of the body found tuberculous. Often in children the glands on the lung root become enlarged and caseous and penetrate deeply into the hilus and into the lung itself.

The symptoms of enlarged mediastinal glands are very uncertain in the simple and tuberculous forms. On the other hand in Hodgkin's disease and in sarcoma pressure symptoms are the rule.

Much attention has been paid ~~recently~~ to the diagnosis of this condition and authors speak quite lightly of the possibility of recognizing by percussion the various grades of enlargement. Indeed, it is claimed by Kronig and others that the pressure of the glands on the right bronchus may cause a dulness in the right lung apex due to slight collapse. The shadows on the X-ray picture cast by enlarged glands are believed to be distinctive, and examined in this way the percentage of cases in children is very high, 50 to 60 in some series.

D'Espine says there is a change in the whispered voice which has a bronchial ring at the level of the seventh cervical and last dorsal, and the respiratory murmur may be rougher and harsher.

Suppurative Lymphadenitis.—Occasionally abscess in the bronchial or

Inspection of nitrogen - carbonic acid process. [illegible]

Repeated tapping.

[illegible]

Chronic meningitis [illegible] - Chronic of Syphilis.

in children - upper or lower limb may be involved. Gradual lessening of [illegible] - [illegible] type rises.

" adults - upper part more often involved - osseous cortex or diaphysis

may be the only local [illegible] of bone or [illegible] - no Syphilis.

Sympathetic [illegible] - [illegible] - 150 [illegible] of the vessels of [illegible]

Haemophysis . not uncommon.

May simulate ulcerous pul. TB

tracheal lymph-glands is found. It may follow the simple adenitis, but is most frequently associated with the presence of tubercle. The liquid portion may gradually become absorbed and the inspissated contents undergo calcification. Serious accidents occasionally occur, as perforation into the œsophagus or into a bronchus, or in rare instances, as in the case reported by Sidney Phillips, perforation of the aorta, as well as a bronchus, which, it is remarkable to say, did not prove fatal rapidly, but caused repeated attacks of hæmoptysis during a period of sixteen months.

Tumors: Cancer and Sarcoma.—In Hare's elaborate study of 520 cases of disease of the mediastinum there were 134 cases of cancer, 98 cases of sarcoma, 21 cases of lymphoma, 7 cases of fibroma, 11 cases of dermoid cysts, 8 cases of hydatid cysts and instances of lipoma, gumma and enchondroma. From this we see that cancer is the most common form of growth. The tumor occurred in the anterior mediastinum alone in 48 of the cases of cancer and in 33 of the cases of sarcoma. There are three chief points of origin, the thymus, the lymph-glands, and the pleura and lung. Sarcoma is more frequently primary than cancer. Males are more frequently affected than females. The age of onset is most commonly between thirty and forty.

Symptoms.—The signs of mediastinal tumor are those of intrathoracic pressure. In some cases almost the entire chest is filled with the masses. The heart and lungs are pushed back and it is marvelous how life can be maintained with such dislocation and compression of the organs. *Dyspnœa* is one of the earliest and most constant symptoms, and may be due either to pressure on the trachea or on the recurrent laryngeal nerves. It may, indeed, be cardiac, due to pressure upon the heart or its vessels. In a few cases it results from the pleural effusion which so frequently accompanies intrathoracic growths. Associated with the dyspnœa is a cough, often severe and paroxysmal in character, with the brazen quality of the so-called aneurismal cough when a recurrent nerve is involved. The voice may also be affected from a similar cause. Pressure on the vessels is common. The superior vena cava may be compressed and obliterated, and when the process goes on slowly the collateral circulation may be completely established. Less commonly the inferior vena cava or one or other of the subclavian veins is compressed. The arteries are much more rarely obstructed. There may be dysphagia, due to compression of the œsophagus. In rare instances there are pupillary changes, either dilatation or contraction, due to involvement of the sympathetic. Expectoration of blood, pus, and hair is characteristic of the dermoid cyst, of which Christian has collected 40 cases.

Physical Signs.—On inspection there may be orthopnœa and marked cyanosis of the upper part of the body. In such instances, if of long duration, there are signs of collateral circulation and the superficial mammary and epigastric veins are enlarged. In these cases of chronic obstruction the finger-tips may be clubbed. There may be bulging of the sternum or the tumor may erode the bone and form a prominent subcutaneous growth. The rapidly growing lymphoid tumors more commonly than others perforate the chest wall. In 4 of 13 cases of Hodgkin's disease there was mediastinal growth, and in 3 instances the sternum was eroded and perforated. The perforation may be on one side of the breast-bone. The projecting tumor may pulsate; the heart may be dislocated and its impulse much out of place. Con-

traction of one side of the thorax has been noted in a few instances. On palpation the fremitus is absent wherever the tumor reaches the chest wall. If pulsating, it rarely has the forcible, heaving impulse of an aneurismal sac. On auscultation there is usually silence over the dull region. The heart-sounds are not transmitted and the respiratory murmur is feeble or inaudible, rarely bronchial. Vocal resonance is, as a rule, absent. Signs of pleural effusion occur in a great many instances of mediastinal growth, and in doubtful cases the aspirator needle should be used.

Tumors of the anterior mediastinum originate usually in the thymus, or its remnants, or in the connective tissue; the sternum is pushed forward and often eroded. The growth may be felt in the suprasternal fossa; the cervical glands are usually involved. The pressure symptoms are chiefly upon the venous trunks. Dyspnœa is a prominent feature.

Intrathoracic tumors in the middle and posterior mediastinum originate most commonly in the lymph-glands. The symptoms are out of all proportion to the ~~physical~~ signs; there is urgent dyspnœa and cough, which is sometimes loud and ringing. The pressure symptoms are chiefly upon the gullet, the recurrent laryngeal, and sometimes upon the azygos vein.

In a third group, tumors originating in the pleura and the lung, the pressure symptoms are not so marked. Pleural exudate is very much more common; the patient becomes anæmic and emaciation is rapid. There may be secondary involvement of the lymph-glands in the neck.

Diagnosis.—The diagnosis of mediastinal tumor from aneurism is sometimes extremely difficult. An interesting case reported and figured by Sokolosski, in Bd. 19 of the Deutsches Archiv für klinische Medicin, in which Oppolzer diagnosed aneurism and Skoda mediastinal tumor, illustrates how in some instances the most skillful of observers may be unable to agree. Scarcely a sign is found in aneurism which may not be duplicated in mediastinal tumor. This is not strange, since the symptoms in both are largely due to pressure. The cyanosis, the venous engorgement, the signs of collateral circulation are, as a rule, much more marked in tumor. The time element is important. If a case has persisted for more than eighteen months the disease is probably aneurism. There are, however, exceptions to this. By far the most valuable sign of aneurism is the diastolic shock so often to be felt, and in a majority of cases to be heard, over the sac. This is rarely, if ever, present in mediastinal growths, even when they perforate the sternum and have communicated pulsation. Tracheal tugging is rarely present in tumor. Another point of importance is that a tumor, advancing from the mediastinum, eroding the sternum, and appearing externally, if aneurismal, has forcible, heaving, and distinctly expansile pulsations. The radiating pain in the back and arms and neck is rather in favor of aneurism, as is also a beneficial influence on it of iodide of potassium. The remarkable traumatic cyanosis of the upper half of the body which follows compression injuries of the thorax could scarcely be mistaken for the effect of tumor. ~~In skillful hands~~ The X-ray picture gives us ~~now a means of~~ differentiating aneurism and tumor ~~which~~ is rarely at fault.

The frequency of pleural effusion in connection with mediastinal tumor is to be constantly borne in mind. It may give curiously complex characters

to the physical signs—characters which are profoundly modified after aspiration of the liquid. Occasionally a tumor of the mediastinum is operable.

Abscess of the Mediastinum.—Hare collected 115 cases of mediastinal abscess, in 77 of which there were details sufficient to permit the analysis. Of these cases the great majority occurred in males. Forty-four were instances of acute abscess. The anterior mediastinum is most commonly the seat of the suppuration. The cases are most frequently associated with trauma. Some have followed erysipelas or occurred in association with eruptive fevers. Many cases, particularly the chronic abscesses, are of tuberculous origin. Of *symptoms,* pain behind the sternum is the most common. It may be of a throbbing character, and in the acute cases is associated with fever, sometimes with chills and sweats. If the abscess is large there may be dyspnœa. The pus may burrow into the abdomen, perforate through an intercostal space, or ~~it may~~ erode the sternum. Instances are on record in which the abscess has discharged into the trachea or œsophagus. In many cases, particularly of chronic abscess, the pus becomes inspissated and produces no ill effect. The *physical signs* may be very indefinite. A pulsating and fluctuating tumor may appear at the border of the sternum or at the sternal notch. The absence of *bruit,* of the diastolic shock, and of the expansile pulsation usually enables a correct diagnosis to be made. When in doubt a fine hypodermic needle may be inserted.

Indurative Mediastino-Pericarditis.—Harris has reviewed the subject. In one form there are adherent pericardium and great increase in the fibrous tissues of the mediastinum; in another there is adherent pericardium with union to surrounding parts, but very little mediastinitis; in a third the pericardium may be uninvolved. The disease is rare; of 22 cases 17 were in males; only 2 were above thirty years of age. The symptoms are essentially those of that form of adhesive pericardium which is associated with great hypertrophy and dilatation of the heart, and in which the patients present a picture of cyanosis, dyspnœa, anasarca, etc. The pulsus paradoxus, described by Kussmaul, is not distinctive. Occasionally there is also a proliferative peritonitis. Mediastinal friction is sometimes heard in patients with adhesive mediastino-pericarditis—dry, coarse, crackling râles heard along the sternum, particularly when the arms are raised.

Miscellaneous Affections.—In Hare's monograph there were 7 instances of fibroma, 11 cases of dermoid cyst, 8 cases of hydatid cyst, and cases of lipoma and gumma.

Emphysema of the Mediastinum.—Air in the cellular tissues of the mediastinum is met with in cases of trauma, and occasionally in fatal cases of diphtheria and in whooping-cough. It may extend to the subcutaneous tissues. Champneys ~~has~~ called attention to its frequency after tracheotomy, in which, he says, the conditions favoring the production are division of the deep fascia, obstruction in the air-passages, and inspiratory efforts. The deep fascia, he says, should not be raised from the trachea. It is often associated with pneumothorax, and more often in rupture of the lung without pneumothorax, the pleura remaining intact and the air dissecting its way along the bronchi into the mediastinum and into the neck. The condition seems by no means uncommon. Angel Money found it in 16 of 28 cases of tracheotomy, and in 2 of these pneumothorax also was present.

SECTION VII

DISEASES OF THE KIDNEYS

I. MALFORMATIONS

Newman classifies the malformations of the kidney as follows: A. Displacements without mobility—(1) congenital displacement without deformity; (2) congenital displacement with deformity; (3) acquired displacements. B. Malformations of the kidney. I. Variations in number—(*a*) supernumerary kidney; (*b*) single kidney, congenital absence of one kidney, atrophy of one kidney; (*c*) absence of both kidneys. II. Variations in form and size—(*a*) general variations in form, lobulation, etc.; (*b*) hypertrophy of one kidney; (*c*) fusion of two kidneys—horseshoe kidney, sigmoid kidney, disk-shaped kidney. C. Variations in pelvis, ureters, and blood-vessels.

The fused kidneys may form a large mass, which is often displaced, being either in an iliac fossa or in the middle line of the abdomen, or even in the pelvis. Under these circumstances it may be mistaken for a new growth. In Polk's case the organ was removed under the belief that it was a floating kidney. The patient lived eleven days, had complete anuria, and it was found post mortem that a single fused kidney had been removed. A second case of the same kind has been reported.

II. MOVABLE KIDNEY

(*Floating Kidney; Palpable Kidney; Ren mobilis; Nephroptosis*)

Known to Riolan in the 17th century and to Matthew Baillie and to Rayer in the first half of the 19th century, it is only during the past quarter of a century that the condition has attracted widespread attention.

The kidney is held in position by its fatty capsule, by the peritoneum which passes in front of it, and by the blood-vessels. Normally the kidney is firmly fixed, but under certain circumstances one or the other organ, more rarely both, becomes movable. In very rare cases the kidney is surrounded, to ~~a greater or less~~ extent, by the peritoneum, and is anchored at the hilus by a mesonephron. Some would limit the term floating kidney to this condition.

Movable kidney is almost always acquired. It is more common in women. Of the 667 cases collected in the literature by Kuttner, 584 were in women and only 83 in men. It is more common on the right than on the left side. Of 727 cases analyzed by this author, it occurred on the right in 553 cases, on

the left in 81, and on both sides in 93. The greater frequency of the condition in women may be attributed to compression of the lower thoracic zone by tight lacing, and, more important still, to the relaxation of the abdominal walls which follows repeated pregnancies. This does not account for all the cases, as movable kidney is by no means uncommon in nulliparæ. In many cases there is a congenitally relaxed condition of the peritoneal attachments. The condition has been met with in infants and in children. Wasting of the fat about the kidney may be a cause. Trauma and the lifting of heavy weights are occasionally factors. The kidney is sometimes dragged down by tumors. The greater frequency on the right side is probably associated with the position of the kidney just beneath the liver, and the depression to which the organ is subjected with each descent of the diaphragm in inspiration.

And, lastly, movable kidney is met with in many cases which present that combination of neurasthenia with gastro-intestinal disturbance which has been described by Glénard as *enteroptosis* (see p. ~~548~~).

To determine the presence of a movable kidney the patient should be placed in the dorsal position, with the head moderately low and the abdominal walls relaxed. The left hand is placed in the lumbar region behind the eleventh and twelfth ribs; the right hand in the hypochondriac region, in the nipple line, just under the edge of the liver. Bimanual palpation may detect the presence of a firm, rounded body just below the edge of the ribs. If nothing can be felt, the patient should be asked to draw a deep breath, when, if the organ is palpable, it is touched by the fingers of the right hand. Various grades of mobility may be recognized. It may be possible barely to feel the lower edge on deep palpation—*palpable kidney*—or the organ may be so far displaced that on drawing the deepest breath the fingers of the right hand may be, in a thin person, slipped above the upper end of the organ, which can be readily held down, but can not be pushed below the level of the navel—*movable kidney.* In a third group of cases the organ is freely movable, and may even be felt just above Poupart's ligament, or may be in the middle line of the abdomen, or can even be pushed over beyond this point. To this the term *floating kidney* is appropriate.

The movable kidney is not painful on pressure, except when it is grasped very firmly, when there is a dull pain, or sometimes a sickening sensation. Examination of the patient from behind may show a distinct flattening in the lumbar region on the side in which the kidney is mobile.

Symptoms.—In a large majority of cases there are no symptoms, and if detected accidentally it is well not to let the patient know of its presence. Far too much stress has been laid upon the condition ~~of late years~~. In other instances there is pain in the lumbar region or a sense of dragging and discomfort, or there may be intercostal neuralgia. In a large group the symptoms are those of neurasthenia with dyspeptic disturbance. In women the hysterical symptoms may be marked, and in men various grades of hypochondriasis; and various forms of insanity have been attributed to it! The gastric disturbance is usually a form of nervous dyspepsia. Dilatation of the stomach has been observed, owing, as suggested by Bartels, to pressure of the dislocated kidney upon the duodenum. The association with a *depressed* stomach is common in women. Constipation is not infrequent. Some writers have described pressure upon the gall-ducts, with jaundice, but this is

very rare. Fæcal accumulation and even obstruction may be associated with the displaced organ.

DIETL'S CRISES.—In connection with movable kidney, nearly always in women, and on the right side, there are remarkable attacks characterized by pain, chill, nausea, vomiting, fever, and collapse. They were described first by Dietl, in 1864, and attributed to twist or kink of the renal vessels or of the ureter. In the subject of movable kidney they may recur at intervals for months or years. A sudden exertion, an error in diet, or standing for a long time may bring on an attack. The pain is in the renal region, of great intensity, simulating colic, and radiates down to the ureter and through to the back. The patient feels nauseated and cold, or there may be a severe chill; vomiting is common. The urine is scanty and contains an excess of urate and oxalates; sometimes it is bloody. Locally there are two conditions, the affected side is tender, the muscular tension increases, and the kidney may be felt enlarged, sensitive to pressure and less movable; but there is no positive tumor. In other cases a tumor rapidly forms from dilatation of the pelvis of the kidney. Appearing, first anteriorly, at the edge of the epigastric region, it may gradually reach the size of a large orange or a cocoanut and fills the entire renal region. This may happen within thirty-six or forty-eight hours. The nausea persists, there is fever, the patient looks ill, and the urine may be scanty or bloody. The general symptoms abate, the local tenderness lessens, the amount of urine may increase rapidly, and in ten or twelve hours the tumor may disappear. In a month or two with a return of the symptoms the tumor re-appears, and again subsides. This is the well-known condition of *intermittent hydronephrosis*, which is one of the most serious and distressing of the sequels of movable kidney.

Diagnosis.—The diagnosis of movable kidney is rarely doubtful, as the shape of the organ is usually distinctive and the mobility marked. Tumors of the gall-bladder, ovarian growths, and tumors of the bowels may in rare instances be confounded with it.

Treatment.—In many instances the greatest relief is experienced from a bandage and pad. It should be applied in the morning, with the patient in the dorsal or knee-breast position, and she should be taught how to push up the kidney. An air pad may be used if the organ is sensitive. In other cases a ~~broad bandage well padded~~ in the lower abdominal zone pushes up the intestines and makes ~~them act as a support~~. In the attacks of severe colic morphia is required. When dependent, as seems sometimes the case, upon an excess of uric acid or the oxalates, the diet must be carefully regulated. The intermittent hydronephrosis may be relieved by the pad and bandage. It rarely demands immediate operation. The kidney may have to be stitched in position.

Stitching of the kidney—nephrorrhaphy—is the most suitable procedure for severe cases, and relief is afforded in many instances by the operation, though not in all. Treatment designed to increase fat-formation often helps to hold the kidney in place. In the neurasthenic cases a prolonged rest treatment is indicated.

has the same effect/

Atheulin shows always the fault in the Sexual nervous condition

III. CIRCULATORY DISTURBANCES

Normally the secretion of urine is accomplished by the maintenance of a certain blood pressure within the glomeruli and by the activity of the renal epithelium. The watery elements are filtered from the glomeruli, the amount depending on the rapidity and the pressure of the blood current; the quality, whether normal or abnormal, depending upon the condition of the capillary and glomerular epithelium; while the greater portion of the solid ingredients are excreted by the epithelium of the convoluted tubules. The integrity of the epithelium covering the capillary tufts within Bowman's capsule is essential to the production of a normal urine. If under any circumstances their nutrition fails, as when, for example, the rapidity of the blood current is lowered, so that they are deprived of the necessary amount of oxygen, the material which filters through is no longer normal (i. e., water), but contains serum albumin. Cohnheim has shown that the renal epithelium is extremely sensitive to circulatory changes, and that compression of the renal artery for only a few minutes causes serious disturbance.

The circulation of the kidney is remarkably influenced by reflex stimuli coming from the skin. Exposure to cold causes heightened blood pressure within the kidneys and increased secretion of urine. Bradford has shown that after excision of portions of the kidney, to as much as one-third of the total weight, there is a remarkable increase in the flow of urine.

Congestion of the Kidneys.—(1) Active Congestion; Hyperæmia.—Acute congestion of the kidney is met with in the early stage of nephritis, whether due to cold or to the action of poisons and severe irritants. Turpentine, cubebs, cantharides, and copaiba cause extreme hyperæmia of the organ. The most typical congestion of the kidney which we see post mortem is that in the early stage of acute Bright's disease, when the organ may be large, nephritis soft, of a dark color, and on section blood drips from it freely.

It has been held that in all the acute fevers the kidneys are congested, and that this explained the scanty, high colored, and often albuminous urine. On the other hand, by Roy's oncometer, Walter Mendelson has shown that the kidney in acute fever is in a state of extreme anæmia, small, pale, and bloodless; and that this anæmia, increasing with the pyrexia and interfering with the nutrition of the glomerular epithelium, accounts for the scanty, dark-colored urine of fever and for the presence of albumin. In the prolonged fevers, however, it is probable that relaxation of the arteries again takes place. Certainly it is rare to find post mortem such a condition of the kidney as is described by Mendelson. On the contrary, the kidney of fever is commonly swollen, the blood-vessels are congested, and the cortex frequently shows traces of cloudy swelling. However, the circulatory disturbances in acute fevers are probably less important than the irritative effects of either the specific agents of the disease or the products produced in their growth or in the altered metabolism of the tissues. The urine is diminished in amount, and may contain albumin and tube-casts, sometimes much of the former and few of the latter.

(2) Passive Congestion; Mechanical Hyperæmia.—This is found in cases of chronic disease of the heart or lung, with impeded circulation, and as

a result of pressure upon the renal veins by tumors, the pregnant uterus, or ascitic fluid. In the cardiac kidney, as it is called, the cyanotic induration associated with chronic heart disease, the organs are enlarged and firm, the capsule strips off, as a rule, readily, the cortex is of a deep red color, and the pyramids of a purple red. The section is coarse looking, the substance is very firm, and resists cutting and tearing. The interstitial tissue is increased, and there is a small-celled infiltration between the tubules. Here and there the Malpighian tufts have become sclerosed. The blood-vessels are usually thickened, and there may be more or less granular, fatty, or hyaline changes in the epithelium of the tubules. The condition is indeed a diffuse nephritis. The urine is usually reduced, is of high specific gravity, and contains more or less albumin. Hyaline tube casts and blood corpuscles are not uncommon. In some cases (over half) with macroscopically no signs of chronic or acute nephritis the urinary features lead to the diagnosis of acute nephritis (Emerson). In uncomplicated cases of the cyanotic induration uræmia is rare. On the other hand, in the cardiac cases with extensive arterio-sclerosis, the kidneys are more involved and the renal function is likely to be disturbed.

IV. ANOMALIES OF THE URINARY SECRETION

1. ANURIA

Total suppression of urine occurs under the following conditions:

(*a*) As an event in the intense congestion of acute nephritis. For a time no urine may be formed; more often the amount is greatly reduced.

(*b*) More commonly complete anuria is seen in subjects of renal stone, fragments of which block both ureters; or as in a case reported by Monod the calculus blocked the only kidney, the other being represented by a shell of tissue. In this "obstructive suppression," as it is called, there is a condition which has been called latent uræmia. There may be very little discomfort, and the symptoms are very unlike those of ordinary uræmia. Convulsions occurred in only 5 of 41 cases (Herter); headache in only 6; vomiting in only 12. Consciousness is retained; the pupils are usually contracted; the temperature may be low; there are twitchings and perhaps occasional vomiting. Of 41 cases in the literature, 35 occurred in males. Of 36 cases in which there was absolute anuria, in 11 the condition lasted more than four days, in 18 cases from seven to fourteen days, and in 7 cases longer than fourteen days (Herter). Obstructive suppression is met with also when cancer compresses both ureters and involves their orifices in the bladder.

(*c*) Cases occur occasionally in which the ~~suppression~~ is prerenal. The following are among the more important conditions with which this form of anuria may be associated: Fevers and inflammations; acute poisoning by phosphorus, lead, and turpentine; in the collapse after severe injuries or after operations, or, indeed, after the passing of a catheter; in the collapse stage of cholera and yellow fever; and, lastly, there is an hysterical anuria, of which Charcot reports a case in which the suppression lasted for eleven days. Bailey reports the case of a young girl, aged eleven, inmate of an orphan asylum, who passed no urine from October 10th to December 12th (when 8

Arctic Chronometer revolving round axis.

Mercury [illegible].

in Arctic Chronometer revolving the round axis;

hypernephroma.
pyelonephritis.
trauma.
hydronephrosis
carcinoma

Bladder - carcinoma papilloma - angioma
tuberculosis. polypi

ounces were withdrawn), and again from this date to March 1st! The question of hysterical deception was considered in the case.

A patient may live for from ten days to two weeks with complete suppression. In Polk's case, in which the only kidney was removed, the patient lived eleven days. It is remarkable that in many instances there are no toxic features. Adams reports a case of recovery after nineteen days of suppression.

In the obstructive cases surgical interference should be resorted to. In the non-obstructive cases, particularly when due to extreme congestion of the kidney, cupping over the loins, hot applications, free purging, and sweating with pilocarpine and hot air are indicated. When the secretion is once started diuretin often acts well. Large hot irrigations, with normal salt solution, with Kemp's double-current rectal tubes, should be tried, as they are stated to stimulate the activity of the kidneys in a remarkable way.

2. HÆMATURIA

Etiology.—The following division may be made of the condition in which hæmaturia occurs:

(1) Essential Hæmaturia.—To this condition much attention has been paid, ~~of late, as the surgeons have taught us to recognize its frequency.~~ The not very happy name of renal hæmophilia has been given to it. In this condition bleeding takes place from one or both of the kidneys without any evidence of disease to the naked eye or to the microscope. Angioma and capillary nævi of the renal papillæ and of the pelves of the kidney are excluded. The subjects are usually under the age of thirty. The bleeding is spontaneous, often associated with pain, though in many cases the attacks are painless. The X-ray picture is negative, the hæmorrhage ceases of itself, and only in a few cases do the attacks recur with such frequency that the patient becomes anæmic. The condition has been referred to under Gull's name of renal epistaxis in ~~several~~ previous editions. It is rarely serious, and many cases recover spontaneously, in others the nephrotomy stops the tendency to bleeding, though why it should do so is difficult to say. The outlook for patients is good (see Hale White, *Q. J. M.*, 1911).

(2) General Diseases.—In the malignant specific fevers, in purpura, and occasionally in leukæmia.

(3) Renal Causes.—Acute congestion and inflammation, as in ~~Bright's disease~~ nephritis, or the effect of toxic agents, such as turpentine, carbolic acid, and cantharides. When the carbolic spray was in use many surgeons suffered from hæmaturia in consequence ~~of this poison~~. Renal infarction, as in ulcerative endocarditis. New growths, in which the bleeding is usually profuse. In tuberculosis at the onset, when the papillæ are involved, there may be bleeding. Stone in the kidney is a frequent cause. Parasites: The *Filaria sanguinis hominis* and the *Bilharzia* cause a form of hæmaturia met with in the tropics. The echinococcus is rarely associated with hæmorrhage. It is sometimes met with in floating kidney, and in pyelonephritis

(4) Affections of the Urinary Passages.—Stone in the ureter, tumor or ulceration of the bladder, the presence of a calculus, parasites, and, very rarely, ruptured veins in the bladder. Bleeding from the urethra occasionally occurs in gonorrhœa and as a result of the lodgment of a calculus. Re- tubercl. polypi diverticula

curring hæmaturia may be an early symptom in enlarged prostate. An unusual cause is the painful, villous tumor of the renal pelvis, of which Savory and Nash report a remarkable case and have collected 49 others from the literature. It would be difficult to recognize the condition from stone. Angioma and capillary nævi of the papillæ may cause bleeding.

(5) Traumatism.—Injuries may produce bleeding from any part of the urinary passages. By a fall or blow on the back the kidney may be ruptured, and this may be followed by very free bleeding; less commonly the blood comes from injury of the bladder or of the prostate. Blood from the urethra is frequently due to injury by the passage of a catheter, or sometimes to falls. Transient hæmaturia follows all operations on the kidney.

The malarial hæmaturia has already been considered.

Diagnosis.—The diagnosis of hæmaturia is usually easy. The color of the urine varies from a light smoky to a bright red, or it may have a dark porter color. Examined with the microscope, the blood-corpuscles are readily recognized, either plainly visible and retaining their color, in which case they are usually crenated, or simply as shadows. In ammoniacal urine or urines of low specific gravity the hæmoglobin is rapidly dissolved from the corpuscles, but in normal urine they remain for many hours unchanged.

It is important to distinguish between blood coming from the bladder and from the kidneys, though this is not always easy. From the bladder the blood may be found only with the last portions of urine, or only at the termination of micturition. In hæmorrhage from the kidneys the blood and urine are intimately mixed. Clots are more commonly found in the blood from the kidneys, and may form moulds of the pelvis or of the ureter. When the seat of the bleeding is in the bladder, on washing out this organ, the water is more or less blood-tinged; but if the source of the bleeding is higher, the water comes away clear. In many instances it is difficult to settle the question by the examination of the urine alone, and the symptoms and the physical signs must also be taken into account. Cystoscopic examination of the bladder, paying especial attention to the urine flowing from each ureteral orifice, and catheterization of the ureters are aids in the diagnosis of doubtful cases.

3. HÆMOGLOBINURIA

This condition is characterized by the presence of blood-pigment in the urine. The blood-cells are either absent or in insignificant numbers. The coloring matter is not hæmatin, as indicated by the old name, *hæmatinuria*, nor in reality always hæmoglobin, but it is most frequently methæmoglobin. The urine has a red or brownish-red, sometimes quite black, color, and usually deposits a very heavy brownish sediment. When the hæmoglobin occurs only in small quantities, it may give a lake or smoky color to the urine. Microscopic examination shows the presence of granular pigment, sometimes fragments of blood disks, epithelium, and very often darkly pigmented urates. The urine is also albuminous. The number of red blood corpuscles bears no proportion whatever to the intensity of the color of the urine. Examined spectroscopically, there are either the two absorption bands of oxyhæmoglobin, which is rare, or, more commonly, there are the three absorption bands of

malignant pustule.

multiple. tubercles.

ulcerative — perforating + tumors
multiple

the study - 738 cases. J.A.M.A. Febr 24. 17
Kretschmer.

1. tumor.
2. tuberculosis
3. new growths
4. calculi.

methæmoglobin, of which the one in the red near *C* is characteristic. Two clinical groups may be distinguished.

Toxic Hæmoglobinuria.—This is caused by poisons which produce rapid dissolution of the blood corpuscles, such as potassium chlorate in large doses, pyrogallic acid, carbolic acid, arseniuretted hydrogen, carbon monoxide, naphthol, and muscarine; also the poisons of scarlet fever, yellow fever, typhoid fever, malaria, and syphilis. It has also followed severe burns. Exposure to excessive cold and violent muscular exertion are stated to produce hæmoglobinuria. A most remarkable toxic form occurs in horses, coming on with great suddenness and associated with paresis of the hind legs. Death may occur in a few hours or a few days. The animals are attacked only after being stalled for some days and then taken out and driven, particularly in cold weather. The form of hæmoglobinuria from cold and exertion is extremely rare. No instance of it, even in association with frost-bites, came under ~~my~~ observation in Canada. Blood transfused from one mammal into another causes dissolution of the corpuscles with the production of hæmoglobinuria; and, lastly, there is the *epidemic hæmoglobinuria* of the newborn, associated with jaundice, cyanosis, and nervous symptoms.

Paroxysmal Hæmoglobinuria.—This rare disease is characterized by the occasional passage of bloody urine, in which the coloring matter only is present. It is more frequent in males than in females, and occurs chiefly in adults. It seems specially associated with cold and exertion, and has often been brought on, in a susceptible person, by the use of a cold foot-bath. It occurs, too, in persons subject to the various forms of Raynaud's disease, and the relation between these two affections is extremely close; some hold that they are manifestations of one and the same disorder. Druitt, the author of the well-known Surgical Vade-mecum, has given a graphic description of his sufferings, which lasted for many years, and were accompanied with local asphyxia and local syncope. The connection, however, is not very common. In only one of the cases of Raynaud's disease which ~~I~~ have seen was paroxysmal hæmoglobinuria present, and ~~in it~~ epileptic attacks occurred at the same time. The relation of hæmoglobinuria to malaria has been considered.

The attacks may come on suddenly after exposure to cold or as a result of mental or bodily exhaustion. They may be preceded by chills and pyrexia. In other instances the temperature is subnormal. There may be vomiting and diarrhœa. Pain in the lumbar region is not uncommon. The hæmoglobinuria rarely persists for more than a day or two—sometimes, indeed, not for a day. There are instances in which, even in ~~the course of~~ a single day, there have been two or three paroxysms, and in the intervals clear urine has been passed. Jaundice has been present in a number of cases. The disease is rarely if ever fatal.

Much has been done latterly to clear up the nature of this remarkable disease by the studies of Eason, Donath, Landsteiner, Hoover and Stone, and Moss. Briefly, the blood serum of these patients contains a complex hæmolysin, a potential toxin, capable of dissolving the patient's own corpuscles and those of other individuals. It is an amboceptor component of the hæmolysin, not the complement, that is peculiar, and this amboceptor differs "from other known hæmolytic amboceptors in that it will unite with the red blood-corpuscles only at a low temperature in the presence of complement, and

furthermore in that it is capable of bringing about the solution of the patient's own cells (auto-hæmolytic action), and those of other members of the group to which the patient belongs, as well as the cells of members of other groups" (Moss). Atmospheric cold and congestion of the peripheral vessels, as in Raynaud's disease, will reduce the temperature of the blood sufficiently to permit of the union of the amboceptor and corpuscles, and hæmolysis occurs when the blood passes to the internal organs.

In a certain number of cases syphilis is present, usually congenital.

Treatment.—In all forms of hæmaturia rest is essential. In that produced by renal calculi the recumbent posture may suffice to check the bleeding. Full doses of acetate of lead and opium should be tried, then calcium lactate, adrenalin, ergot, gallic acid and tannic acid, and the dilute sulphuric acid. The oil of turpentine, sometimes recommended, is a risky remedy. ~~in hæmaturia. Extr. hamamelis virgin. and extr. hydrastis canad. are also recommended.~~ Cold may be applied to the loins or dry cups in the lumbar region. Incision of the kidney has cured the so-called "renal epistaxis."

The treatment of paroxysmal hæmoglobinuria is unsatisfactory. Amyl nitrite will sometimes cut short or prevent an attack (Chvostek). During the paroxysm the patient should be kept warm and given hot drinks. If there is a syphilitic history, iodide of potassium in full doses may be tried. In a warm climate the attacks are much less frequent. It is possible that an antitoxin may be obtained to neutralize the hæmolytic amboceptor of the disease.

4. ALBUMINURIA

"Reasons drawn from the urine are as brittle as the urinal" is a dictum of Thomas Fuller peculiarly appropriate in connection with this subject.

The presence of albumin in the urine, formerly regarded as indicative of ~~Bright's disease~~, is now recognized as occurring under many circumstances without the existence of serious organic change in the kidney. Two groups of cases may be recognized—those in which the kidneys show no coarse lesions, and those in which there are evident anatomical changes.

Albuminuria without Coarse Renal Lesions.—(*a*) FUNCTIONAL, SO-CALLED PHYSIOLOGICAL ALBUMINURIA.—In a normal condition of the kidney only the water and the salts are allowed to pass from the blood. When albuminous substances transude there is probably disturbance in the nutrition of the epithelium of the capillaries of the tuft, or of the cells surrounding the glomerulus. This statement is still, however, in dispute, and many hold that there is a physiological albuminuria which may follow muscular work, the ingestion of food rich in albumin, violent emotions, cold bathing, and dyspepsia. On one point all agree, that the cause must be something unusual and excessive, as a very hard tramp, a football match, a race, etc. The presence of albumin in the urine, in any form and under any circumstance, may be regarded as indicative of change in the renal or glomerular epithelium, a change, however, which may be transient, slight, and unimportant, depending upon variations in the circulation or upon the irritating effects of substances taken with the food or temporarily present, as in febrile states.

Albuminuria of adolescence and cyclic albuminuria, in which the albumin is present only at certain times during the day—orthostatic albuminuria—

essential
The Treatment must depend on the cause.

are interesting forms. A majority of the cases occur in young persons—boys more commonly than girls—and the condition is often discovered accidentally. These are often the children of neurotic parents, and have well-marked vasomotor instability. Some cases last only during puberty, some throughout life. The condition is very common, particularly in young men in training—the athletic albuminuria to which Collier has called attention. Of 156 men in training 130 had albumin in the urine. Erlanger and Hooker have shown that the albumin is excreted only during periods with low pulse pressure. The urine, as a rule, contains only a very small amount of albumin, but in some instances large quantities are present. The most striking feature is the variability. It may be absent in the morning and present only after exertion; or it may be greatly increased after taking food, particularly proteins. Even the change to the upright position (orthostatic) may suffice to cause it, and in such cases there may be tension on the renal veins by increase of the lumbar curve, since it has been shown that a spinal jacket will prevent the appearance of the albumin. The quantity of urine may be but little, if at all, increased, the specific gravity is usually normal, and the color may be high. Occasionally hyaline casts may be found, and in some instances there has been transient glycosuria. As a rule, the pulse is not of high tension and the second aortic sound is not accentuated.

Various forms of this affection have been recognized by writers, such as neurotic, dietetic, cyclic, intermittent, and paroxysmal—names which indicate the characters of the different varieties.

Goodhart, from a study of the after history of more than 250 cases, holds that albuminuria of the adolescent has no sinister effect on health or upon duration of life, and that with due circumspection such cases ought not to be excluded from the advantages of life insurance, ~~or from clerkships in banks and private offices~~. This is a very important ~~and gratifying~~ statement from a man who has made a special study of the subject.

In a few cases the albumin is persistent, the amount is larger, though it may vary from day to day, ~~and~~ the pulse tension is increased, and there are probably indications of organic changes in the kidney.

(*b*) Febrile Albuminuria.—Pyrexia, by whatever cause produced, may cause slight albuminuria. The presence of the albumin is due to slight changes in the glomeruli induced by the fever, such as cloudy swelling, which can not be regarded as an organic lesion. It is extremely common, occurring in pneumonia (in about 70 per cent. of our cases), diphtheria, typhoid fever (about 60 per cent. of our cases), malaria, especially the æstivo-autumnal type, and even in the fever of acute tonsillitis. The amount of albumin is slight, and it usually disappears from the urine with the cessation of the fever. Hyaline and even epithelial casts accompany the condition.

(*c*) Hæmic Changes.—Purpura, scurvy, chronic poisoning by lead or mercury, syphilis, leukæmia, and profound anæmia may be associated with slight albuminuria. Abnormal ingredients in the blood, such as bile pigment and sugar, may cause the passage of small amounts of albumin.

The transient albuminuria of pregnancy may belong to this hæmic group, although in a majority of such cases there are changes in the renal tissue. Albumin may be found sometimes after the inhalation of ether or chloroform.

(*d*) Nervous System.—Under many morbid conditions of the nervous

system, albumin may be present in the urine, and there are instances in young nervous persons which are not easy to separate from the so-called orthostatic forms. In brain tumors, following epileptic attacks, in various types of meningitis, albumin has been present. In meningeal hæmorrhage, as pointed out by Guillain, the albumin may be very abundant, 5 to 20 grams in the litre.

Albuminuria with Definite Lesions of the Urinary Organs.—(*a*) Congestion of the kidney, either active, such as follows exposure to cold and is associated with the early stages of nephritis, or passive, due to obstructed outflow in disease of the heart or lungs, or to pressure on the renal veins by the pregnant uterus or tumors.

(*b*) Organic disease of the kidneys—acute and chronic ~~Bright's disease~~, amyloid and fatty degeneration, suppurative nephritis, and tumors.

(*c*) Affections of the pelvis, ureters, and bladder, when associated with the formation of pus.

Albumosuria.—Albumose, peptone, and globulin are occasionally found in the urine, but are of very slight clinical significance. They are found in many febrile diseases, in chronic suppuration, and whenever protein materials are undergoing autolysis, as in pneumonia, acute yellow atrophy, and during the involution of the uterus.

Myelopathic albumosuria, *"Kahler's disease,"* is characterized by multiple myelomata with persistent excretion of what is known as the Bence-Jones body, a protein discovered by him in 1848. ~~There are now many cases on record~~. Males above forty years of age are usually affected. The Bence-Jones body appears rarely with other tumors of the bones. The myeloma is a true tumor, the cells of which resemble the plasma rather than the myelocytes of the bone marrow (Christian). In a case ~~which I saw~~ with Hamburger the persistent albumosuria led to the diagnosis of multiple myelomata before any bone tumors could be felt. The disease runs a fatal course. The simplest reaction is the white precipitate formed on adding nitric acid to the urine; when boiled it disappears, to reappear on cooling. As in one of Bradshaw's cases, the urine may be of a milky white color when passed.

Prognosis.—Febrile albuminuria is transient, and in a majority of the cases depending upon hæmic causes the condition disappears and leaves the kidneys intact. A trace of albumin in a man over forty, with or without a few hyaline casts, is not of much significance, except as an indication that his kidneys, like his hair, are beginning to turn "gray" with age. In many instances the discovery is a positive advantage, as the man is made to realize, perhaps for the first time, that he has been living carelessly. ~~I have~~ discussed the question from this standpoint in a paper with the paradoxical title "On the Advantages of a Trace of Albumin and a Few Tube-casts in the Urine of Men over Fifty Years of Age" (*N. Y. Med. Jour.,* vol. lxxiv).

The persistence of a slight amount of albumin in young men without increased arterial tension is less serious, as even after continuing for years it may disappear. The outlook in the so-called cyclic albuminuria has been discussed.

Practically in all cases the presence of albumin indicates a change of some sort in the glomeruli, the nature, extent, and gravity of which it is difficult to estimate; so that other considerations, such as the presence of

prostate

and prostate

Bence-Jones — somewhat in nature of protease but higher protein of
endogenous origin — derived from tumor cells of
myeloma or some abnormal synthesis of normal protein.

B-J. protein has power of sensitization + anaphylactic response in animals
this suggests that it higher protein. than a protease.
This protein is excreted by a kidney which holds back normal serum proteins
maybe a product of tumor cells of myeloma
or normal or aberrant stage in synthesis of some body protein.

H. Cabot. Surg. Gyn. Obst. Nov. 16.

renal affection. - if proteus mirabilis shows cocci in abundance - Scale ends of cells
1. few s.b.c. + many bacteria or a bacillus - + renal
function near normal = coccus affection.
2. if many bacilli. little alb. + much pus with renal
diminished function = colon bacillus.

① mostly in cortex. + slightly in convoluted tubes.
② first in convoluted tubule. - pelvis lesion Sect 1-11 ①.

tube-casts, the existence of increased tension, the general condition of the patient, and the influence of digestion upon the albumin, must be carefully considered.

The physician is daily consulted as to the relation of albuminuria and life assurance. As his function is to protect the interests of the company, he should reject all cases in which albumin occurs in the urine, except in young persons with transient albuminuria. Naturally, companies lay great stress upon the presence or absence of albumin, but in the most serious and fatal malady with which they have to deal—chronic interstitial nephritis—the albumin is often absent or transient, even when the disease is well developed. After the fortieth year, from a standpoint of life insurance, the state of the arteries and the blood pressure are far more important than the condition of the urine.

5. BACTERIURIA

Described first by Roberts in 1881, much attention has been paid to it of late years, and its importance recognized both as a secondary and a primary affection. The secondary form is best illustrated by the common bacilluria of typhoid fever, already described. In the cases in which there is no recognizable cause or primary focus of the disease, the colon bacillus, streptococci, and the gonococcus are the commonest organisms. The bacilli may come directly from the blood, as in typhoid fever, and probably multiply in the urinary passages, or they may come from a focus of infection anywhere from Bowman's capsule to the prostate.

Clinically there are two groups of cases, the bacilluria pure and simple and the bacilluric cystitis or pyelitis. In the former there may be no symptoms; the urine may have a slight haziness due to the enormous number of organisms, but there is no pus. In the other there are signs of inflammatory reaction in the urinary passages and there is pus. Usually with the *Bacillus coli* infection the urine is acid, with the staphylococcus alkaline and often with marked phosphaturia. The cases are often very intractable. Without cystitis or pyelitis there may be no symptoms, but in too many instances there are all the aggravated phenomena of these two affections. Many cases clear up rapidly with hexamethylenamine. Vaccine therapy has been extensively used but not with very good results.

6. PYURIA

(*Pus in the Urine*)

Causes.—(*a*) Pyelitis and Pyelonephritis.—In large abscesses of the kidney, pyonephrosis, the pus may be intermittent, while in calculus and tuberculous pyelitis the pyuria is usually continuous, though varying in intensity. In cases due to the colon or tubercle bacillus the urine is acid, in those due to the proteus bacillus alkaline, while in the staphylococcus cases the urine is either less acid than normal, or alkaline. In the pyelitis and pyelonephritis following cystitis the urine is alkaline or acid, depending upon the infecting micro-organism; more mucus, frequent micturition, and a previous bladder history are aids in diagnosis.

(*b*) CYSTITIS.—The urine is usually acid, especially in women, since the colon bacillus is a very common cause of these infections. The pus and mucus are more ropy, and triple phosphate crystals are found in the freshly passed urine in the alkaline infections.

(*c*) URETHRITIS, particularly gonorrhœa. The pus appears first, is in small quantities, and there are signs of local inflammation.

(*d*) In LEUCORRHŒA the quantity of pus is usually small, and large flakes of vaginal epithelium are numerous. In doubtful cases, when leucorrhœa is present, the urine should be withdrawn through a catheter.

(*e*) RUPTURE OF ABSCESSES INTO THE URINARY PASSAGES.—In such cases as pelvic or perityphlitic abscess there have been previous symptoms of pus formation. A large amount is passed within a short time, then the discharge stops abruptly or rapidly diminishes within a few days.

Pus gives to the urine a white or yellowish-white appearance. On settling, the sediment is sometimes ropy, the supernatant fluid usually turbid. In cases due to urea-decomposing microbes (proteus bacillus, various staphylococci) the odor may be ammoniacal even in fresh urine. Examination with the microscope reveals the presence of a large number of pus-corpuscles, which are usually, when the pus comes from the bladder, well formed; the protoplasm is granular, and often shows many translucent processes.

The only sediment likely to be confounded with pus is that of the phosphates; but it is whiter and less dense, and is distinguished immediately by microscopic examination or by the addition of acid.

With the pus there is always more or less epithelium from the bladder and pelves of the kidneys, but since in these situations the forms of cells are practically identical, they afford no information as to the locality from which the pus has come.

The treatment of pus in the urine is considered under the conditions in which it occurs.

7. CHYLURIA—NON-PARASITIC

This is a rare affection, occurring in temperate regions and unassociated with the *Filaria bancrofti*. The urine is of an opaque white color; it resembles milk closely, is occasionally mixed with blood (hæmatochyluria), and sometimes coagulates into a firm, jelly-like mass. In other instances there is at the bottom of the vessel a loose clot which may be distinctly blood tinged. Under the microscope the turbidity seems to be caused by numerous minute granules—more rarely oil droplets similar to those of milk. In Montreal I made the dissection in a case of thirteen years' duration and could find no trace of parasites. The urine may be much more milky shortly after taking food, and the recumbent posture increases the milkiness. It has been shown in one case that the urine only became chylous in the bladder, and Hertz found obstruction of the thoracic duct and a communicating ruptured lymphatic vessel in the bladder.

8. LITHURIA

The general relations of uric acid have already been considered in speaking of gout.

Occurrence in the Urine.—The uric acid occurs in combination chiefly

fish &c
no meat. tea, coffee.

with ammonium and sodium, forming the acid urates. In smaller quantities are the potassium, calcium, and lithium salts. The uric acid may be separated from its bases and crystallizes in rhombs or prisms, which are usually of a deep red color, owing to the staining of the urinary pigments. The sediment formed is granular and the groups of crystals look like grains of Cayenne pepper. It is very important not to mistake a deposit of uric acid for an excess. The deposition of numerous grains in the urine within a few hours after passing is more likely to be due to conditions which diminish the solvent power than to increase in the quantity. Of the conditions which cause precipitation of the uric acid Roberts gives the following: "(1) High acidity; (2) poverty in mineral salts; (3) low pigmentation; and (4) high percentage of uric acid." The grade of acidity is probably the most important element.

In health the weight of uric acid excreted bears a fairly constant ratio to the weight of urea eliminated. According to von Noorden, the average ratio is 1 to 50, while the average ratio of the nitrogen of uric acid to the total nitrogen eliminated in the urine is 1 to 70. In several ~~of the~~ cases of gout ~~in my wards~~ Futcher found that in the intervals between the acute arthritic attacks the uric acid was reduced to a much greater extent than the urea, so that the ratio of the former to the latter often varied between 1 to 300 up to (in one case) 1 to 1,500, a return to about the normal proportions occurring during the acute attacks.

More common is the precipitation of amorphous urates, forming the so-called brick-dust or lateritious deposit, which has a pinkish color, due to the presence of urinary pigment. It is composed chiefly of the acid sodium urates. It occurs particularly in very acid urine of a high specific gravity. As the urates are more soluble in warm solutions, they frequently deposit as the urine cools. Here, too, the deposition does not necessarily, indeed usually does not, mean an excessive excretion, but the existence of conditions favoring the deposit.

9. OXALURIA

The discovery of calcium oxalate crystals in the urine by Donné in 1838 led to the description of the so-called oxalic-acid diathesis. It is claimed that all the oxalic acid found in the urine is taken into the body with the food (Dunlop). In health none, or only a trace, is formed in the body. The amount fluctuates with the quantity of food taken, and is usually below 10 milligrams daily (H. Baldwin). It seems to be formed in the body when there is an absence of free hydrochloric acid in the gastric juice, and in connection with excessive fermentation in the intestines. It never forms a heavy deposit, but the crystals—usually octahedral, rarely dumb-bell-shaped—collect in the mucous cloud and on the sides of the vessel.

When in excess and present for any considerable time, the condition is known as oxaluria, the chief interest of which is in the fact that the crystals may be deposited before the urine is voided, and form a calculus. It is held by many that there is a special diathesis associated with its presence in excess and manifested clinically by dyspepsia, particularly the nervous form, irritability, depression of spirits, lassitude, and sometimes marked hypochondriasis. There may be in addition neuralgic pains and the general symp-

toms of neurasthenia. The local and general symptoms are probably dependent upon some disturbance of metabolism of which the oxaluria is one of the manifestations. It is a feature also in many gouty persons, and in the condition called lithæmia.

10. CYSTINURIA

This rare condition, a sort of chemical malformation (Garrod), is of clinical importance because cystin is very sparingly soluble and calculi may be formed, renal or vesical. It is strongly hereditary and has been traced through three generations. The quantity excreted is about 0.5 gram per diem, and the excretion persists for years, or even for life, without causing disturbance of health. Cystin is one of the primary fractions of protein, and its excretion is one of the as yet unexplained errors of protein metabolism. In the urinary sediment the colorless hexagonal crystals of cystin are readily detected.

11. PHOSPHATURIA

The phosphoric acid is excreted from the body in combination with potassium, sodium, calcium, and magnesium, forming two classes, the alkaline phosphates of sodium and potassium and the earthy phosphates of lime and magnesia. The amount of phosphoric acid (P_2O_5) excreted in the twenty-four hours varies, according to Hammarsten, between 1 and 5 grams, with an average of 2.5 grams. It is derived mainly from the phosphoric acid taken in the food, but also in part as a decomposition product from nuclein, protagon, and lecithin. Of the alkaline phosphates, those in combination with sodium are the most abundant. The alkaline phosphates of the urine are more abundant than the earthy phosphates.

Of the *earthy phosphates,* those of lime are abundant, of magnesium scanty. In urine which has undergone the ammoniacal fermentation, either inside or outside the body, there is in addition the ammonio-magnesium or triple phosphate, which occurs in triangular prisms or in feathery or stellate crystals; hence the term of stellar phosphates given to this form. The earthy phosphates occur as a sediment in the urine when the alkalinity is due to a fixed alkali, or under certain circumstances the deposit may take place within the bladder, and then the phosphates are passed at the end of micturition as a whitish fluid, which is popularly confounded with spermatorrhœa. Recent study of these cases with symptoms of neurasthenia and a phosphate sediment in the fresh urine would indicate an abnormality in the calcium metabolism, an absolute increase of this with a decrease of the phosphoric acid. The calcium phosphate may be precipitated by heat and produce a cloudiness which may be mistaken for albumin, but is at once dissolved upon making the urine acid. This condition is very frequent in persons suffering from dyspepsia or from debility of any kind. The phosphates may be in great excess, rising in the twenty-four hours to from 7 to 9 grams (Teissier), whereas the normal amount is not more than 2.5 grams. And, lastly, the phosphates may be deposited in urine which has undergone decomposition, in which the carbonate of ammonia from the urea combines with the magnesium phosphates, forming the triple salt. This is seen in *cystitis,* due to a urea decomposing microbe.

The clinical significance of an excess of phosphates, to which the term

Upon ... use of the ... acid ... [illegible] ... [illegible] ...

Tr. decrease amt. of cystine & increase solubility in urine.

Can decrease by cutting off protein. Then only endogenous cystine

in urine 70 mg. per day.

Alkali - helps solubility 6-10 gm. sod. bicarb. a day. Clean clear

phosphaturia is applied, has been much discussed. It must be remembered that a deposit does not necessarily mean an excess, to determine which a careful analysis of the twenty four hours' secretion should be made. It has long been thought that there is a relation between the activity of the nerve tissues and the output of phosphoric acid; but the question can not yet be considered settled. The amount is increased in wasting diseases, such as ~~phthi~~tuber-sis, acute yellow atrophy of the liver, leukæmia, and severe anæmia, whereas it is diminished in acute diseases and during pregnancy.

Teissier, of Lyons, in 1876, described a condition to which he gave the name of "essential phosphaturia," and it has been called "phosphatic diabetes," the symptoms of which are polyuria, thirst, emaciation, and a great increase in the excretion of phosphates, which would rise to as much as 7 to 9 grams a day. The condition sometimes simulates true diabetes very closely, even to the pruritis and dry skin. In a remarkable case of this kind, under ~~my~~ observation for several years, Barker studied the metabolism ~~very thoroughly~~, and found it normal for carbohydrates, but the organic phosphorus percentage was high; the chief abnormality, ~~however~~, was an ~~abnormally~~ unusually large amount of organic acids, so that chemically the condition was suggestive of an acidosis.

12. INDICANURIA

The substance in the urine which has received this name is the indoxyl-sulphate of potassium, in which form it appears in the urine and is colorless. When concentrated acids or strong oxidizing agents are added to the urine, this substance is decomposed and the indigo set free. It is present only in small quantities in healthy urine. It is derived from the indol, a product formed in the intestine by the decomposition of the albumin under the influence of bacteria. When absorbed, this is oxidized in the tissues to indoxyl, which combines with the potassium sulphate, forming ~~the above named substance~~ indican.

It is a common condition met with accidentally in persons of good health or with slight digestive complaints. It is not specially associated with constipation (Allen Jones). In gall-stone attacks, in hyperchlorhydria, in recurring appendicitis, in wasting diseases, in peritonitis, and in empyema it is usually present. In a few cases it is constantly present and in excess. ~~In a recent study~~ Barr found only 32 such cases among 2,092 patients, and in these the clinical symptoms did not suggest an intestinal auto-intoxication, nor did the lacto-bacillary treatment of Metschnikoff have the slightest influence on the condition.

Indican has occasionally been found in calculi. Though, as a rule, the urine is colorless when passed, there are instances in which the decomposition has taken place within the body, and a blue color has been noticed immediately after the urine was voided. Sometimes, too, in alkaline urine on exposure there is a bluish film on the surface. Methylene blue, a coloring matter for candy, etc., must be excluded.

13. MELANURIA

Black urine may be dark when passed or may become so later. In the following conditions melanuria may occur: (1) Jaundice. Only in very

chronic cases of deeply bronzed icterus do we see the urine quite dark, due to the presence of large quantities of biliverdin. (2) Hæmaturia and hæmoglobinuria. Here it is a matter of the exaggeration of the smoky tint due to the presence of blood in various quantities. (3) Hæmatoporphyrinuria, to be considered later. (4) Melanuria, in which the urine has, as a rule, the normal color when passed, and on standing becomes black as ink. In some instances it is black when passed. Melanuria of this type only occurs with the presence of melanotic tumors. (5) Alkaptonuria. (6) Indicanuria. When rich in indoxyl sulphate the urine is brown in color, or becomes so after standing, due to the oxidation products of indol. This is by far the most common cause of black urine, and in any disease leading to an abundant secretion of indican, as in intestinal obstruction, etc., black urine may be passed. As Garrod suggests, it is probable that the black urine in cases of tuberculosis is of an allied nature. (7) After certain articles of diet and drugs. Some dark colored vegetable pigments, as black cherries and plums and bilberries, cause darkening of the urine. Resorcin may do the same. Carboluria is by no means uncommon, and was frequently seen in the days of the antiseptic spray. It has been ascribed to hydrochinone formed from phenol. Naphthalene, creosote, and the salicylates may cause darkening of the urine, or even blackness.

14. ALKAPTONURIA

"Alkaptonuria is not the manifestation of a disease, but is rather of the nature of an alternative course of metabolism, harmless and usually congenital and lifelong" (Garrod). Of 40 ~~known~~ examples, 19 occurred in seven families, and several were the offspring of first cousins (Garrod). There are two points of clinical interest. The alkapton urine reduces Fehling's solution, and diabetes may be suggested, but it does not ferment, and it is optically inactive. The linen may be stained by the urine, which in some cases is dark when passed. In 1866 Virchow recorded a case of blackening of the cartilages and ligaments—ochronosis, which is considered elsewhere.

15. PNEUMATURIA

Gas may be passed with the urine—

1. After mechanical introduction of air in vesical irrigation or cystoscopic examination in the knee elbow position.

2. As a result of the introduction of gas forming organisms in catheterization or other operation. Glycosuria has been present in a majority of the cases. The yeast fungus, the colon bacillus, and the *Bacillus aërogenes capsulatus* have been found.

3. In cases of vesico-enteric fistula.

In gas production within the bladder the symptoms are those of a mild cystitis, with the passage of gas at the end of micturition, sometimes with a loud sound. The diagnosis is readily made by causing the patient to urinate in a bath or by plunging the end of the catheter under water.

Renal Function

Creatinin - in blood - often increased in uraemia. a rise above 2.5 mg. per 100 cc. is a means renal involvement. 2.5 - 3 mg. is suspicious - 3 - 5 mg. is unfavorable - above 5 mg. nearly death.

normally very constant. 1 - 2 mg.

Urea - (Strength). N - protein nitrogen divided

Excessive permeability of kidney.
to water, salts, albumen etc.

In convulsions. Toxin has isolated toxicbase.

In uraemia of arthritis. Urea usually not more than 100 mg. per 100 cc. blood,
marked symptoms of 200 mg. per 100 cc.
Uraemia (symptoms if over 100 mg. per 100 c.c.)

16. OTHER SUBSTANCES

Lipuria.—Fat in the urine, or lipuria, occurs, first, without disease of the kidneys, as in excess of fat in the food, after the administration of cod liver oil, in fat embolism occurring after fractures, in the fatty degeneration in phosphorus poisoning, in prolonged suppuration, as in phthisis and pyæmia, in the lipæmia of diabetes mellitus; secondly, with disease of the kidneys, as in the fatty stage of chronic Bright's disease, in which fat casts are sometimes present, and, according to Ebstein, in pyonephrosis; and, thirdly, in the affection known as chyluria. The urine is usually turbid, but there may be fat drops as well, and fatty crystals have been found. In a few rare instances calculi composed of fat and coated with phosphates have been found.

Lipaciduria is a term applied by von Jaksch to the condition in which there are volatile fatty acids in the urine, such as acetic, butyric, formic, and propionic acid.

The occurrence of *acetone*, *diacetic acid*, and *β-oxybutyric acid* has been sufficiently considered under Diabetes.

Choluria and **glycosuria** have already been considered under jaundice and diabetes.

Hæmatoporphyrin occasionally occurs in the urine. It was first recognized by Hoppe-Seyler. Nencki and Sieler determined its exact formula, and the former demonstrated that the only chemical difference between hæmatin and hæmatoporphyrin is that the latter is simply hæmatin free from iron. It has been found in the urine in pulmonary tuberculosis, pleurisy with effusion, acute rheumatism, lead poisoning, and intestinal hæmorrhages. This pigment has been found very frequently after the administration of sulphonal, and sometimes imparts a very dark color to the urine.

V. URÆMIA

Definition.—A toxæmia developing in the course of nephritis or in conditions associated with anuria. The nature of the poison or poisons is as yet unknown, whether they are the retained normal products or the products of an abnormal metabolism.

Theories of Uræmia.—There are four chief views: (*a*) That it is due to the accumulation in the blood of body poisons which should be excreted by the kidney. What these substances are is not known, but neither the urea, nor the salts, nor the various extractives appear to be capable of producing the symptoms. (*b*) That it is a disturbance of the normal kidney metabolism. Brown-Séquard suggested that the kidney had an internal secretion, to the disturbance of which it is thought that the symptoms of uræmia may be due. Rose Bradford's experiments show how profoundly the kidneys influence the body metabolism, particularly that of the muscles. If more than two-thirds of the total kidney weight is removed, there is an extraordinary increase in the production of urea and of the nitrogenous bodies of the creatin class. From a study of the question Hughes and Carter conclude that the poison is of an albuminous nature, and quite different from anything in normal urine. (*c*) Uræmia has been attributed to nephro-

lysins. Broken up renal substance contains a material which, when injected into an animal, exerts a specific destructive action upon the renal cells. This substance may be found in the serum, and such a blood serum injected into another animal sets up nephritis and albuminuria. It is suggested that the phenomena of uræmia may result from the action of these nephrolysins upon the nerve centres. F. Müller has pointed out that uræmia is most apt to occur in those forms of renal disease in which the destruction of tissue is most extensive. (*d*) Lastly, there is the old view of Traube that the symptoms of uræmia, particularly the coma and convulsions, are due to localized œdema of the brain.

Symptoms.—Clinically, we may recognize latent, acute, and chronic forms. The latent form has been considered under the section on anuria. Acute uræmia may arise in any form of nephritis. It is more common in the post-febrile varieties. Bradford thinks that it is specially associated with a form of contracted white kidney in young subjects. Chronic forms of uræmia are more frequent in the arterio-sclerotic and granular kidney. For convenience the symptoms of uræmia may be described under cerebral, dyspnœic, and gastro-intestinal manifestations.

Among the CEREBRAL symptoms of uræmia may be described:

(*a*) *Mania.*—This may come on abruptly in an individual who has shown no previous indications of mental trouble, and who may not be known to have ~~Bright's disease~~ nephritis. In one case of this kind the patient became suddenly maniacal and died in six days. More commonly the delirium is less violent, but the patient is noisy, talkative, restless, and sleepless.

(*b*) *Delusional Insanity* (*Folie Brightique*).—Cases are by no means uncommon, and excellent clinical reports have been issued on the subject from several of the asylums, particularly by Bremer, Christian, and Alice Bennett. Delusions of persecution are common. ~~One of my patients committed suicide by jumping out of a window.~~ The condition is of interest medico-legally because of its bearing on testamentary capacity. Profound melancholia may also supervene.

(*c*) *Convulsions.*—These may come on unexpectedly or be preceded by pain in the head and restlessness. The attacks may be general and identical with those of ordinary epilepsy, though the initial cry may not be present. The fits may recur rapidly, and in the interval the patient is usually unconscious. Sometimes the temperature is elevated, but more frequently it is depressed, and may sink rapidly after the attack. Local or Jacksonian epilepsy may occur in most characteristic form in uræmia. A remarkable sequence of the convulsions is blindness—*uræmic amaurosis*—which may persist for several days. This, however, may occur apart from the convulsions. It usually passes off in a day or two. There are, as a rule, no ophthalmoscopic changes. Sometimes uræmic deafness supervenes, and is probably also a cerebral manifestation. It may also occur in connection with persistent headache, nausea, and other gastric symptoms.

(*d*) *Coma.*—Unconsciousness invariably accompanies the general convulsions, but a coma may develop gradually without any convulsive seizures. Frequently it is preceded by headache, and the patient gradually becomes dull and apathetic. In these cases there may have been no previous indications of renal disease, and unless the urine is examined the nature of the

Nobl. Lec. from back. pressure - Ky. [illegible]
here drainage. [illegible]
removed [illegible]

1. retention type
2. renal or edema type
3. toxic type or [illegible]

Uraemia - Types. Hosler J.A.M.A. - Sept 23. 16.

1. simple retention of urinary nitrogenous waste - urinary poisons
2. defective water + salt metabolism - results in renal edema
3. Toxemia - resulting from abnormal katabolism.

2 and 3 or [illegible] - N. waste [illegible]
3rd. may show also those 3 [illegible] by an abnormal process in
normal katabolism + resulting in toxemia - shown clinically
convulsive seizures. Why rarely do we see types uncomplicated

[illegible]

excellent?

Hubert Wakeman.
Jour. Exp. Med. 1899 - IV

case may be overlooked. Twitchings of the muscles occur, particularly in the face and hands, but there are many cases of coma in which the muscles are not involved. In some of these cases a condition of torpor persists for weeks or even months. The tongue is usually furred and the breath very foul and heavy.

(*e*) *Local Palsies.*—In the course of chronic ~~Bright's disease~~ nephritis hemiplegia or monoplegia may come on spontaneously or follow a convulsion, and post mortem no gross lesions of the brain be found, but only a localized or diffused œdema. These cases, which are not very uncommon, may simulate almost every form of organic paralysis of cerebral origin.

(*f*) Of other cerebral symptoms, HEADACHE is important. It is most often occipital and extends to the neck. It may be an early feature and associated with giddiness. Other nervous symptoms of uræmia are intense itching of the skin, numbness and tingling in the fingers, and cramps in the muscles of the calves, particularly at night. An erythema may be present.

URÆMIC DYSPNŒA is classified by Palmer Howard as follows: (*a*) Continuous dyspnœa; (*b*) paroxysmal dyspnœa; (*c*) both types alternating; and (*d*) Cheyne-Stokes breathing. The attacks of dyspnœa are most commonly nocturnal; the patient may sit up, gasp for breath, and evince as much distress as in true asthma. Occasionally the breathing is noisy and stridulous. The Cheyne-Stokes type may persist for weeks or months. One patient, up and about, could feed himself only in the apnœa period. Though usually of serious omen and occurring with coma and other symptoms, recovery may follow even after persistence for a long period.

The GASTRO-INTESTINAL manifestations of uræmia often set in with abruptness. Uncontrollable vomiting may come on and its cause be quite unrecognizable. The attacks may be preceded by nausea and may be associated with diarrhœa. In some instances the diarrhœa may come on without the vomiting; sometimes it is profuse and associated with an intense catarrhal or even diphtheritic inflammation of the colon.

A special URÆMIC STOMATITIS has been described in which the mucosa of the lips, gums, and tongue is swollen and erythematous. The saliva may be increased, and there is difficulty in swallowing and in mastication. The tongue is usually very foul and the breath heavy and fetid. A cutaneous erythema may occur and a remarkable urea "frost" on the skin.

FEVER is not uncommon in uræmic states, and may occur with the acute nephritis, with the complications, and as a manifestation of the uræmia itself.

Very many patients with chronic uræmia succumb to ~~what I have called~~ terminal infections—acute peritonitis, pericarditis, pleurisy, meningitis, or endocarditis.

Diagnosis.—Herter called attention to the value of the clinical determination of the urea in the blood (for which purpose only a few cubic centimetres are required) as an index of the degree of renal inadequacy. In but 2 of 96 cases could the urea determination in the urine have been of any value in predicting uræmia, and equal drops in the urea occurred without this symptom (Emerson). The test of the functional capacity of the kidney by the use of phenol-sulphonephthalein (Rowntree and Geraghty) is of great value both in differential diagnosis and in giving warning of impending uræmia. In uræmia the elimination of phthalein is nil or only a faint trace in two hours. In

patients with chronic nephritis in whom the elimination in two hours is below 10 per cent. there is grave danger of uræmia.

Uræmia may be confounded with:

(*a*) Cerebral lesions, such as hæmorrhage, meningitis, or even tumor. In apoplexy, which is so commonly associated with kidney disease and stiff arteries, the sudden loss of consciousness, particularly if with convulsions, may simulate a uræmic attack; but the mode of onset, the existence of complete hemiplegia, with conjugate deviation of the eyes, suggest hæmorrhage. As already noted, there are cases of uræmic hemiplegia or monoplegia which can not be separated from those of organic lesion and which post mortem show no trace of coarse disease of the brain. Indeed, in some of these cases it is quite impossible to distinguish between the two conditions. So, too, cases of meningitis, in a condition of deep coma, with perhaps slight fever, furred tongue, but without localizing symptoms, may readily be confounded with uræmia.

(*b*) With certain infectious diseases. Uræmia may persist for weeks or months and the patient lies in a condition of torpor or even unconsciousness, with a heavily coated, perhaps dry, tongue, muscular twitchings, a rapid feeble pulse, with slight fever. This state not unnaturally suggests the existence of one of the infectious diseases. Cases of the kind are not uncommon, and I have known them to be mistaken for typhoid fever and for miliary tuberculosis.

(*c*) Uræmic coma may be confounded with poisoning by alcohol or opium. In opium poisoning the pupils are contracted; in alcoholism they are more commonly dilated. In uræmia they are not constant; they may be either widely dilated or of medium size. The examination of the eye grounds should be made to determine the presence or absence of albuminuric retinitis. The urine should be drawn off and examined. The odor of the breath sometimes gives an important hint.

The condition of the heart and arteries should also be taken into account. Sudden uræmic coma is more common in the chronic interstitial nephritis. The character of the delirium in alcoholism is sometimes important, and the coma is not so deep as in uræmia or opium poisoning. It may for a time be impossible to determine whether the condition is due to uræmia, profound alcoholism, or hæmorrhage into the pons Varolii.

And, lastly, in connection with sudden coma, it is to be remembered that insensibility may occur after prolonged muscular exertion, as after running a ten-mile race. In some instances unconsciousness has come on rapidly with stertorous breathing and dilated pupils. Cases have occurred under conditions in which sun-stroke could be excluded; and Poore considers that the condition is due to the too rapid accumulation of waste products in the blood, and to hyperpyrexia from suspension of sweating.

The treatment will be considered under Chronic Bright's Disease.

VI. ACUTE BRIGHT'S DISEASE

Definition.—Acute diffuse nephritis, due to the action of cold or of toxic agents upon the kidneys.

Dec 7

—

Organisms often found in cancer.

—

many due to chicken - often from tumors.

Cancer of stomach due to [illegible]

In all instances changes exist in the epithelial, vascular, and intertubular tissues, which vary in intensity in different forms; hence writers have described a tubular, a glomerular, and an acute interstitial nephritis. Delafield recognizes *acute exudative* and *acute productive* forms, the latter characterized by proliferation of the connective-tissue stroma and of the cells of the Malpighian tufts.

Etiology.—The following are the principal causes of acute nephritis:

(1) Cold. Exposure to cold and wet is one of the most common causes. It is particularly prone to follow exposure after a drinking-bout.

(2) The poisons of the specific fevers, particularly scarlet fever, less commonly typhoid fever, measles, diphtheria, small-pox, chicken-pox, malaria, cholera, yellow fever, meningitis, and, very rarely, dysentery. Acute nephritis may be associated with syphilis and with acute tuberculosis, particularly the former. Bradford suggests that many of the idiopathic cases and those ascribed to cold may be of syphilitic origin. It may also occur in septicæmia and in acute tonsillitis. In exudative erythema and the allied purpuric affections acute nephritis is not uncommon. Among 1,832 cases of malaria at the Johns Hopkins Hospital there were 26 of nephritis (Thayer). A primary infective epidemic nephritis has been met with in Italy.

(3) Toxic agents, such as turpentine, cantharides, potassium chlorate, and carbolic acid, may cause an acute congestion which sometimes terminates in nephritis. Alcohol probably never excites an acute nephritis.

(4) Pregnancy, in which the condition is probably due to toxic products as yet undetermined.

(5) Acute nephritis occurs occasionally in connection with extensive lesions of the skin, as in burns or in chronic skin-diseases, and also after trauma. It may follow operations on the kidney.

Morbid Anatomy.—The kidneys may present to the naked eye in mild cases no evident alterations. When seen early in more severe forms the organs are congested, swollen, dark, and on section may drip blood. Bright's original description is as follows:

"The kidneys, . . . stripped easily out of their investing membrane, were large and less firm than they often are, of the darkest chocolate color, interspersed with a few white points, and a great number nearly black; and this, with a little tinge of red in parts, gave the appearance of a polished fine-grained porphyry or greenstone. . . . On (section) these colors were found to pervade the whole cortical part; but the natural striated appearance was not lost, and the external part of each mass of tubuli was particularly dark . . . a very considerable quantity of blood oozed from the kidney, showing a most unusual accumulation in the organ."

In other instances the surface is pale and mottled, the capsule strips off readily, and the cortex is swollen, turbid, and of a grayish red color, while the pyramids have an intense beefy red tint. The glomeruli in some instances stand out plainly, being deeply swollen and congested; in other instances they are pale.

The histology may be thus summarized: (*a*) *Glomerular changes.* The tufts suffer first, and there is either an acute intracapillary glomerulitis, in which the capillaries become filled with cells and thrombi, or involvement of

the epithelium of the tuft and of Bowman's capsule, the cavity of which contains leucocytes and red blood-corpuscles.

(*b*) The *alterations in the tubular epithelium* consist in cloudy swelling, fatty change, and hyaline degeneration. In the convoluted tubules, the accumulation of altered cells with leucocytes and blood-corpuscles causes the enlargement and swelling of the organ.

(*c*) *Interstitial changes.* In the milder forms a simple inflammatory exudate—serum mixed with leucocytes and red blood-corpuscles—exists between the tubules. In severer cases areas of small celled infiltration occur about the capsules and between the convoluted tubes.

Symptoms.—The onset is usually sudden, and, when the nephritis follows cold, dropsy may be noticed within twenty-four hours. After fevers the onset is less abrupt, but the patient gradually becomes pale and a puffiness of the face or swelling of the ankles is first noticed. In children there may be convulsions at the outset. Chilliness or rigors initiate the attack in a limited number of cases. Pain in the back, nausea, and vomiting may be present. The fever is variable. Many cases in adults have no rise in temperature. In young children with nephritis from cold or scarlet fever the temperature may, for a few days, range from 101° to 103°.

The most characteristic symptoms are the *urinary changes.* There may at first be suppression; more commonly the urine is scanty, highly colored, and contains blood, albumin, and tube casts. The quantity is reduced and only 4 or 5 ounces may be passed in the twenty-four hours; the specific gravity is high—1.025, or even more; the color varies from a smoky to a deep porter color, but is seldom bright red. On standing there is a heavy deposit; microscopically there are blood corpuscles, epithelium from the urinary passages, and hyaline, blood, and epithelial tube casts. The albumin is abundant, forming a curdy, thick precipitate. The largest amounts of albumin are seen in the early acute nephritis of syphilis, in which it may reach 8.5 per cent. The total excretion of urea is reduced, though the percentage is high. *Uræmia* is an early and marked symptom. In cases of extensive dropsy effusion may take place into the pleuræ and peritoneum. There are cases of scarlatinal nephritis in which the dropsy of the extremities is trivial and effusion into the pleuræ extensive. The lungs may become œdematous. In rare cases there is œdema of the glottis. Epistaxis may occur or cutaneous ecchymoses may develop in the course of the disease.

The pulse may be hard, the tension increased, and the second sound in the aortic area accentuated. Occasionally dilatation of the heart comes on rapidly and may cause sudden death. The skin is dry and it may be difficult to induce sweating.

Uræmic symptoms occur in a limited number of cases, either at the onset with suppression, more commonly later in the disease. Ocular changes are not so common in acute as in chronic ~~Bright's disease~~, but hæmorrhagic retinitis may occur and occasionally papillitis.

The course ~~of acute Bright's disease~~ varies considerably. The description just given is of the form which most commonly follows cold or scarlet fever. In many of the febrile cases dropsy is not a prominent symptom, and the diagnosis rests rather with the examination of the urine. Moreover, the condition may be transient and less serious. In other cases, as in the acute

Acute exacerbations in chronic nephritis.

Acute exacerbations in chronic nephritis are often regarded as primary acute attacks. The condition of the heart & vessels, the study of the blood pressure and of the eye grounds are important points in ~~that diagnosis~~ recognizing this.

nephritis of typhoid fever, there may be hæmaturia and pronounced signs of interference with the renal function. The most intense acute nephritis may exist without anasarca.

In scarlatinal nephritis, in which the glomeruli are most seriously affected, suppression of the urine may be an early symptom, the dropsy is apt to be extreme, and uræmic manifestations are common. Acute ~~Bright's disease~~ nephritis in children, however, may set in very insidiously and be associated with transient or slight œdema, and the symptoms may point rather to affection of the digestive system or to brain disease.

Diagnosis.—It is very important to bear in mind that the most serious involvement of the kidneys may be manifested only by slight œdema of the feet or puffiness of the eyelids, without impairment of the general health. On the other hand, from the urine alone a diagnosis can not be made with certainty, since simple cloudy swelling, and circulatory changes may cause a similar condition of urine. The first indication of trouble may be a uræmic convulsion. This is particularly the case in the acute nephritis of pregnancy, and it is a good rule for the practitioner, when engaged to attend a case, invariably to ask that during the seventh and eighth months the urine should occasionally be sent for examination.

In nephritis from cold and in scarlet fever the symptoms are usually marked and the diagnosis is rarely in doubt. As already mentioned, every case in which albumin is present should not be called acute ~~Bright's disease~~, nephritis, not even if tube casts be present. Thus the common febrile albuminuria, although it represents the first link in the chain of events leading to acute ~~Bright's disease~~, should not be placed in the same category.

There are occasional cases of acute ~~Bright's~~ disease with anasarca, in which albumin is either absent or present only as a trace. This is a rare condition. Tube casts are usually found, and the absence of albumin is rarely permanent. The urine may be reduced in amount.

The character of the casts is of use in the diagnosis of the form of ~~Bright's disease~~, but scarcely of such extreme value as has been stated. Thus, the hyaline and granular casts are common to all varieties. The blood and epithelial casts, particularly those made up of leucocytes, are most common in the acute cases.

Prognosis.—The outlook varies somewhat with the cause ~~of the disease~~. Recoveries in the form following exposure to cold are much more frequent than after scarlatinal nephritis. In younger children the mortality is high, amounting to at least one-third of the cases. Serious symptoms are low arterial tension, the occurrence of uræmia, and effusion into the serous sacs. The persistence of the dropsy after the first month, intense pallor, and a large amount of albumin indicate the possibility of the disease becoming chronic. For some months after the disappearance of the dropsy there may be traces of albumin and a few tube casts.

In a case of scarlatinal nephritis, if the progress is favorable, the dropsy diminishes in a week or ten days, the urine increases, the albumin lessens, and by the end of a month the dropsy has disappeared and the urine is nearly free. In very young children the course may be rapid, and I have known the urine to be free from albumin in the fourth week. Other cases are more insidious, and though the dropsy may disappear, the albumin persists in the

urine, the anæmia is marked, and the condition becomes chronic, or, after several recurrences of the dropsy, improves and complete recovery takes place.

Treatment.—The patient should be in bed and there remain until all traces of the disease have disappeared. As sweating plays such an important part in the treatment, it is well, if possible, to accustom the patient to blankets. He should also be clad in thin Canton flannel.

The diet should consist of milk or butter-milk, gruels made of arrow-root or oat-meal, barley water, and, if necessary, beef tea and chicken broth. It is better, if possible, to confine the patient to a strictly milk diet. As convalescence is established, bread and butter, lettuce, water cress, grapes, oranges, and other fruits may be given. Meats should be used very sparingly. As there is marked retention of the chlorides, which seem to bear a relation to the dropsy, salt should be withheld.

The patient should drink freely of alkaline mineral waters, ordinary water, or lemonade. The fluids keep the kidneys flushed and wash out the *débris* from the tubes. A useful drink is a drachm of cream of tartar in a pint of boiling water, to which may be added the juice of half a lemon and a little sugar. Taken when cold, this is a pleasant ~~and satisfactory~~ diluent drink. Fluid may be given by the bowel or by saline infusion if it is not well taken by mouth.

No remedies, so far as known, control directly the changes which are going on in the kidneys. The indications are: (1) To give the excretory function of the kidney rest by utilizing the skin and the bowels, in the hope that the natural processes may be sufficient to effect a cure; (2) to meet the symptoms as they arise.

In a case of scarlet fever it may occasionally be possible to avert an attack, the premonitory symptoms of which are marked increase in the arterial tension and the presence of blood coloring matter in the urine (Mahomed). An active saline cathartic may completely relieve this condition.

At the onset, when there is pain in the back or hæmaturia, the Paquelin cautery or the dry ~~or wet~~ cups give relief. ~~The last should not be used in children.~~ Warm poultices are often grateful. In cases which set in with suppression of urine these measures should be adopted, and in addition the hot bath with subsequent pack, copious diluents, and a free purge. The dropsy is best treated by hydrotherapy—either the hot bath, the wet pack, or the hot-air bath. In children the wet pack is usually satisfactory. It is applied by wringing a blanket out of hot water, wrapping the child in it, covering this with a dry blanket, and then with a rubber cloth. In this the child may remain for an hour. It may be repeated daily. In the case of adults, the hot air bath or the vapor bath may be conveniently given by allowing the vapor or air to pass from a funnel beneath the bed clothes, which are raised on a low cradle. More efficient, as a rule, is a hot bath of from fifteen or twenty minutes, after which the patient is wrapped in blankets. The sweating produced by these measures is usually profuse, rarely exhausting, and in a majority of cases the dropsy can in this way be relieved. There are some cases, ~~however,~~ in which the skin does not respond to the baths, and if the symptoms are serious, particularly if uræmia supervenes, jaborandi or ~~its active principle,~~ pilocarpine, may be used. The latter may be given hypodermically, in doses of from a sixth to an eighth of a grain (0.01 to 0.008

gm.) in adults, and from a twentieth to a twelfth of a grain (0.003 to 0.005 gm.) in children of from two to ten years.

The bowels should be kept open by a morning saline purge; in children the fluid magnesia is readily taken; in adults the sulphate of magnesia may be given by Hay's method, in concentrated form, in the morning, before anything is taken into the stomach. In ~~Bright's disease~~ it not infrequently causes vomiting. The compound powder of jalap (gr. xx, 1.3 gm.) or, if necessary, elaterium may be used. If the dropsy is not extreme, the urine not very concentrated, and uræmic symptoms are not present, the bowels should be kept loose without active purgation. If these measures fail to reduce the dropsy and it has become extreme, the skin may be punctured ~~with a lancet~~ or drained by a small silver cannula (Southey's tube), which is inserted beneath it. A fine aspirator needle may be used, and the fluid allowed to drain through a piece of long, narrow rubber tubing into a vessel beneath the bed. If the dyspnœa is marked, owing to pressure of fluid in the pleuræ, aspiration should be performed. In rare instances the ascites is extreme and may require paracentesis, or a Southey's tube may be inserted and the fluid gradually withdrawn. If uræmic convulsions occur, the intensity of the paroxysms may be limited by the use of chloroform; to an adult a pilocarpine injection should be at once given, and from a robust, strong man 20 ounces of blood may be withdrawn. In children the loins may be dry cupped, the wet pack used, and a brisk purgative given. Bromide of potassium and chloral sometimes prove useful.

Vomiting may be relieved by ice and by restricting the amount of food. ~~Drop doses of creosote, iodine, and carbolic acid may be given. The dilute hydrocyanic acid with bismuth is often effectual.~~

The question of the use of diuretics in acute ~~Bright's disease~~ is not ~~yet~~ settled. The best diuretic, after all, is water, which may be taken freely with the citrate of potash or the benzoate of soda, salts which are held to favor the conversion of the urates into less irritating and more easily excreted compounds. Digitalis and strophanthus are useful diuretics, and may be employed without risk when the arterial tension is low and the cardiac impulse is not forcible. ~~I have never seen any injurious effects from their employment after the early symptoms had lessened in intensity.~~

For the persistent albuminuria, ~~I agree with Roberts and Rosenstein that~~ we have no remedy of the slightest value. Nothing indicates more clearly our helplessness in controlling kidney metabolism than inability to meet this ~~common~~ symptom. Astringents, alkalies, nitroglycerin, and mercury have been recommended.

For the anæmia associated ~~with acute Bright's disease~~ iron should be employed. ~~It should not be given~~ until the acute symptoms have subsided. In the adult it may be used in the form of the perchloride in increasing doses, as convalescence proceeds. In children, the syrup of the iodide of iron or the syrup of the phosphate of iron are better preparations. Tyson ~~has recently~~ urged caution in the too free use of iron in kidney disease. The dilatation of the heart is best treated with digitalis, strophanthus, and strychnia.

In ~~the~~ convalescence ~~from acute Bright's disease~~ care should be taken to guard the patient against cold. The diet should still consist chiefly of milk

and a return to mixed food should be gradual. A change of air is often beneficial, particularly a residence in a warm, equable climate.

VII. CHRONIC ~~BRIGHT'S DISEASE~~ nephritis

Here, too, in all forms we deal with a diffuse process, involving epithelial, interstitial, and glomerular tissues. Clinically two groups are recognized—(*a*) the chronic parenchymatous nephritis, which follows the acute attack or comes on insidiously, is characterized by marked dropsy, and post mortem by the *large white kidney.* In the later stages of this process the kidney may be smaller—a condition known as the *small white kidney;* (*b*) chronic interstitial nephritis, in which dropsy is not common and the cardio-vascular changes are pronounced. Delafield recognizes a chronic diffuse nephritis with exudation and a chronic productive diffuse nephritis without exudation, the latter corresponding to the contracted kidney of authors.

The amyloid kidney is usually spoken of as a variety of Bright's disease, but in reality it is a degeneration which may accompany any form of nephritis.

1. CHRONIC PARENCHYMATOUS NEPHRITIS

(*Chronic Desquamative and Chronic Tubal Nephritis; Chronic Diffuse Nephritis with Exudation*)

Etiology.—In many cases the disease follows the acute nephritis of cold, scarlet fever, or pregnancy. More frequently than is usually stated the disease has an insidious onset and occurs independently of any acute attack. The fevers may play an important rôle in certain of these cases. Rosenstein, Bartels, I. E. Atkinson, and Thayer have laid special stress upon malaria as a cause. The use of alcohol is believed to lead to this form of nephritis. In chronic suppuration, syphilis, and tuberculosis the diffuse parenchymatous nephritis is not uncommon, and is usually associated with amyloid disease. Males are rather more subject to the affection than females. It is met with most commonly in young adults, and is by no means infrequent in children as a sequence of scarlatinal nephritis.

Morbid Anatomy.—Several varieties of this form have been recognized. The *large white kidney* of Wilks, in which the organ is enlarged, the capsule is thin, and the surface white with the stellate veins injected, is not very common in America. On section the cortex is swollen and yellowish white in color, and often presents opaque areas. The pyramids may be deeply congested. On microscopic examination it is seen that the epithelium is granular and fatty, and the tubules of the cortex are distended, and contain tube casts. Hyaline changes are also present in the epithelial cells. The glomeruli are large, the capsules thickened, the capillaries show hyaline changes, and the epithelium of the tuft and of the capsule is extensively altered. The interstitial tissue is everywhere increased, though not to an extreme degree.

The second variety ~~of this form~~ results from ~~the~~ gradual increase in the connective tissue and ~~the~~ subsequent shrinkage, forming what is called the *small white kidney* or the pale granular kidney. It is doubtful whether this is always preceded by the large white kidney. Some observers hold that it

Camphin. nephritis ; antérieur bradycardie hypertension.

may be a primary independent form. The capsule is thickened and the surface is rough and granular. On section the resistance is greatly increased, the cortex is reduced and presents numerous opaque white or whitish yellow foci, consisting of accumulations of fatty epithelium in the convoluted tubules. This combination of contracted kidney with the areas of marked fatty degeneration has given the name of small granular fatty kidney to this form. The interstitial changes are marked, many of the glomeruli are destroyed, the degeneration of epithelium in the convoluted tubules is widespread, and the arteries are greatly thickened.

Belonging to this chronic tubal nephritis is a variety known as the *chronic hæmorrhagic nephritis*, in which the organs are enlarged, yellowish white in color, and in the cortex are many brownish red areas, due to hæmorrhage into and about the tubes. In other respects the changes are identical with those in the large white kidney.

Of changes in the other organs the most marked are thickening of the blood vessels and hypertrophy of the left heart.

Symptoms.—Following an acute nephritis, the disease may present, in a modified way, the symptoms of that affection. In many cases it sets in insidiously, and after an attack of dyspepsia or a period of failing health and loss of strength the patient becomes pale, and puffiness of the eyelids or swollen feet are noticed in the morning.

The symptoms are as follows: The urine is, as a rule, diminished in quantity, averaging 500 c. c., ~~often scanty~~. It has a dirty yellow, sometimes smoky, color, and is turbid from the presence of urates. On standing, a heavy sediment falls, in which are found numerous tube casts of various forms and sizes, hyaline, both large and small, epithelial, granular, and fatty casts. Leucocytes are abundant; red blood-corpuscles are frequently met with, and epithelium from the kidneys and pelves. The albumin is abundant and may be from 4 to 6 per cent. It is more abundant in the urine passed during the day. The specific gravity may be high in the early stages—from 1.020 to 1.025, even 1.040—though in the later stages it is lower. The urea is always reduced in quantity. As the case improves from 5 to 6 litres of urine a day may be voided.

Dropsy is a marked and obstinate symptom of this form, of ~~Bright's disease~~ nephritis. The face is pale and puffy, and in the morning the eyelids are œdematous. The anasarca is general, and there may be involvement of the serous sacs. In these chronic cases associated with large white kidney there is often a distinctive appearance in the face; the complexion is pasty, the pallor marked, and the eyelids are œdematous. The dropsy is peculiarly obstinate. Uræmic symptoms are common, though convulsions are perhaps less frequent than in the interstitial nephritis.

The tension of the pulse is usually increased; the vessels ultimately become stiff and the heart hypertrophied, though there are instances of this form of nephritis in which the heart is not enlarged. The aortic second sound is accentuated. Retinal changes, though less frequent than in the chronic interstitial nephritis, occur in a considerable number of cases.

Gastro-intestinal symptoms are common. Vomiting is frequently a distressing and serious symptom, and diarrhœa may be profuse. Ulceration of the colon may occur and prove fatal.

It is sometimes impossible to determine, even by the most careful examination of the urine or by analysis of the symptoms, whether the condition of the kidney is that of the large white or of the small white form. In cases, however, which have lasted for several years, with the progressive increase in the renal connective tissue and the cardio-vascular changes, the clinical picture may approach, in certain respects, that of the contracted kidney. The urine is increased, with low specific gravity. It is often turbid, may contain traces of blood, the tube casts are numerous and of every variety of form and size, and the albumin is abundant. Dropsy is usually present, though not so extensive as in the early stages.

Prognosis.—The prognosis is extremely grave. In a case which has persisted for more than a year recovery rarely takes place. Death is caused either by great effusion with œdema of the lungs, by uræmia, or by secondary inflammation of the serous membranes. Occasionally in children, even when the disease has persisted for two years, the symptoms disappear and recovery takes place.

Treatment.—Essentially the same treatment should be carried out as in acute Bright's disease. Milk or buttermilk should constitute for a time the chief article of food. Later more food may be allowed, oysters, fresh vegetables, and fruit. The dropsy should be treated by hot baths, and a salt-free diet. Iron preparations should be given when there is marked anæmia. It is to be remembered that the pallor of the face may not be a good index of the blood condition. The acetate of potash, digitalis, and diuretin are useful in increasing the flow of urine. Basham's mixture given in plenty of water will be found beneficial.

2. CHRONIC INTERSTITIAL NEPHRITIS

(*Secondary Contracted Kidney; Red Granular Kidney; Gouty Kidney; Arterio-sclerotic Kidney; Senile Kidney*)

Etiology and Morbid Anatomy.—Sclerosis of the kidney is met with (*a*) as a sequence of the large white kidney, forming the so-called pale granular or secondary contracted kidney; (*b*) as a primary independent affection, the red granular kidney; (*c*) as a sequence of arterio-sclerosis; and (*d*) as a senile change.

(*a*) SECONDARY FORM.—The small white kidney, as it is called, has already been described as a sequel to chronic parenchymatous nephritis.

(*b*) In the PRIMARY FORM, known also as the red granular kidney, the organ is smaller than in the secondary interstitial nephritis, the capsule is very adherent, the granulations small, the organ of a reddish brown color, the cysts numerous, the arteries very sclerotic, and the cortex greatly reduced in volume. The chief reason for calling this primary is that one can find no history of previous renal disease. It is met with in the members of gouty families, and there are doubtless hereditary influences at work, for Dickinson reported a remarkable family in which this interstitial nephritis occurred in four generations. Syphilis, alcohol, and overeating are mentioned as contributary causes. Lead is a rare cause in America, but a more common cause in parts of England. It is by no means always easy to differentiate

The disease is really of the whole cardiovascular
and system.

between the secondary and the primary forms. As a rule, the former is paler and not so small. Of 174 cases of chronic interstitial nephritis from my wards which came to autopsy, in 79 the combined weight of the kidneys was about 300 grams, in 57 cases 200 to 300 grams, in 30 cases 150 to 200 grams, and below 150 grams in 8 cases (Emerson). Unilateral nephritis in my experience is extremely rare, not occurring once in the series.

(*c*) ARTERIO-SCLEROTIC KIDNEY.—This is not necessarily a contracted kidney. The organ is very hard, red, and often heavier than normal. Of the cases from my wards, studied by Emerson, in 61 per cent. the combined weight was above 300 grams, and in only 6 per cent. was it below 200 grams. The surface may be smooth or the capsule only slightly thickened and adherent, tearing the substance very little as it is stripped off. In other cases the atrophy is in spots, affecting certain vascular districts, so that there is a large, sunken, deep red patch on the surface, or one pole of the kidney is shrunken, or the process is general in both kidneys, but the resulting contraction gives a warty rather than a granular surface.

(*d*) In the SENILE FORM, met with in the aged, the organs are reduced in size, the capsules thickened and adherent, the pelvic fat much increased, both cortical and pyramidal portions uniformly wasted, and the arteries of the kidney substance very prominent.

Almost invariably associated with chronic interstitial nephritis are general arterio-sclerosis and hypertrophy of the heart. The changes in the arteries will be described elsewhere. In the red granular kidney the left ventricle is specially hypertrophied, but in all forms the heart is greatly enlarged, constituting one of the largest forms met with. In many cases the disease is latent, and the patients die of apoplexy or of acute uræmia. In the arterio-sclerotic form death is more commonly cardiac, and the condition of the kidneys may be entirely overlooked.

Much discussion has taken place as to the association of hypertrophy of the heart and sclerosis of the blood-vessels with the renal changes. A complete solution of the many problems has scarcely yet been offered. Briefly, there are two views—the mechanical and the chemical. Dating from the time of Bright it was thought that the heart had greater difficulty in driving the impure blood through the capillary system. Traube held that the obliteration of a large number of capillary territories in the kidney raised the arterial pressure and in this way led to hypertrophy of the heart. In explanation of the muscular hypertrophy of the walls of the smaller arteries George Johnson introduced the view of a stop-cock action of these vessels under the influence of irritating ingredients in the blood. The mechanical view was thus put by Cohnheim. The activity of the circulation through the kidneys at any moment does not depend upon the need of these organs for blood, but solely upon the amount of material for the urinary secretion existing in the blood. When parts of both kidneys have undergone atrophy, the blood flow in the parts remaining must be as great as it would have been to the whole of the organs, had they been intact; but in order that such a quantity of blood should pass through the restricted capillary area now open to it an excessive pressure is necessary. This can be brought to bear only by the exertion of an increased force on the part of the left ventricle with the maintenance of a corresponding resistance in all other arterial territories. In this

way both the high arterial pressure and the cardio-vascular changes are explained.

The chemical view, ~~which has been much discussed of late~~, supposes the production (*a*) by the kidneys, (*b*) by the supra-renal glands, of certain pressor substances. So far as the kidney is concerned, the observations are by no means in accord. Practically we know only that the kidney does contain substances capable of raising the blood-pressure. According to Bingel those so-called rennin preparations act in a manner very different from adrenalin. In chronic interstitial nephritis there is often hyperplasia of the cortical substance of the supra-renals, and many recent writers have claimed to have discovered in the blood of chronic nephritics an increase in the pressor substances, an adrenalinæmia. Through their influence, from one or both of these sources, the blood-pressure is raised, with the inevitable sequence of hypertrophy of the heart and sclerosis of the arteries. As already mentioned, the question is still under discussion.

Symptoms.—Many cases are latent, and are not recognized until the occurrence of one of the serious or fatal complications. Even an advanced grade of contracted kidney may be compatible with great mental and bodily activity. There may have been no symptoms whatever to suggest to the patient the existence of a serious malady. In other cases the general health is disturbed. The patient complains of lassitude, is sleepless, has to get up at night to micturate; the digestion is disordered, the tongue is furred; there are complaints of headache, failing vision, and breathlessness on exertion.

So complex and varied is the clinical picture of ~~chronic Bright's disease~~ that it will be best to consider the symptoms under the various systems.

Urinary System.—In the *small contracted kidney* polyuria is common. Frequently the patient has to get up two or three times during the night to empty the bladder, and there is increased thirst. It is for these symptoms occasionally that relief is sought. And yet in many cases with very small kidneys this feature has not been present. A careful study of the cases ~~from my wards, of the urine and the anatomical condition,~~ showed that almost no parallelism could be made between the weight of the kidney, its appearance, and the urine it secreted before death. Of ~~the~~ 174 cases with autopsy, in almost a third the renal changes were so slight that the nephritis was not mentioned as a part of the clinical diagnosis (Emerson). The color of the urine is a light yellow, and the specific gravity ranges from 1.005 to 1.012. Persistent low specific gravity is one of the most constant and important features of the disease. Traces of albumin are found, but may be absent at times, particularly in the early morning urine. It is often simply a slight cloudiness, and may be apparent only with the more delicate tests. The sediment is scanty, and in it a few hyaline or granular casts are found. The quantity of the solid constituents of the urine is, as a rule, diminished, though in some instances the urea may be excreted in full amount. In attacks of dyspepsia or bronchitis, or in the later stages when the heart fails, the quantity of albumin may be greatly increased and the urine diminished. Occasionally blood occurs in the urine, and there may ~~even~~ be hæmaturia (S. West). Slight leakage, represented by the constant presence of a few red cells, may be present early in the disease and persist for years. In the *arterio-sclerotic form* the quantity of urine is normal, or reduced rather than increased; the specific gravity

Tileston. Arch. Int. Med.
1914. XIV. 620

Healthy adult.

non protein nitrogen - 22.5 - 25 mg. per 100 cc. blood.
urea " 12 - 14 " " " "

both forms nephritis without uraemia. - nor. of urea elevated
uraemic cases - quantities in both
N. of urea

diag. uraemia. usually marked increase - non-prot. N.

if retention in neph. - renal insufficiency.

—

Creatin - Myers & Langh. Arch. Int. Med. Oct. '15. p. 536

high values in blood - in uraemia

over 2.5 mg. per 100 cc. blood always nephritis
over 3.0 cc. "
3 to 5 " " "
over 5

is normal or high, the color of the urine is good, and there are hyaline and finely granular casts. The amount of albumin varies greatly with the food and exercise, and is usually much in excess of that seen with the contracted kidneys, and does not show so often the albumin free intervals of that form, also it is more common to find albumin without casts, while in the contracted kidney casts may occur without albumin.

Circulatory System.—The pulse is hard, the tension increased, and the vessel wall, as a rule, thickened. ~~As already mentioned,~~ a distinction must be made between increased tension and thickening of the arterial wall. The tension may be plus in a normal vessel, but in chronic ~~Bright's disease~~ nephritis it is more common to have increased tension in a stiff artery.

A pulse of increased tension has the following characters: It is hard and incompressible, requiring a good deal of force to overcome it; it is persistent, and in the intervals between the beats the vessel feels full and can be rolled beneath the finger. These characters may be present in a vessel the walls of which are little, if at all, increased in thickness. To estimate the latter the pulse wave should be obliterated in the radial, and the vessel wall felt beyond it. In a perfectly normal vessel the arterial coats, under these circumstances, can not be differentiated from the surrounding tissue; whereas, if thickened, the vessel can be rolled beneath the finger. Persistent high blood pressure is one of the earliest and most important symptoms of interstitial nephritis. During the disease the pressure may rise to 250 mm. or even 300 mm. With dropsy and cardiac dilatation the pressure may fall, but not necessarily. The cardiac features are equally important, though often less obvious. Hypertrophy of the left ventricle occurs to overcome the resistance offered in the arteries. The enlargement of the heart ultimately becomes more general. The apex is displaced downward and to the left; the impulse is forcible and may be heaving. In elderly persons with emphysema the displacement of the apex may not be evident. The first sound at the apex may be duplicated; more commonly the second sound at the aortic cartilage is accentuated, a very characteristic sign of increased tension. The sound in extreme cases may have a bell-like quality. In many cases a systolic murmur develops at the apex, probably as a result of relative insufficiency. It may be loud and transmitted to the axilla. Finally the hypertrophy fails, the heart becomes dilated, gallop rhythm is present, and the general condition is that of a chronic heart-lesion. In the arterio-sclerotic form the picture may be cardiac from beginning to close—dyspnœa and signs of dilated heart.

Respiratory System.—Sudden œdema of the glottis may occur. Effusion into the pleuræ or sudden œdema of the lungs may prove fatal. Acute pleurisy and pneumonia are not uncommon. Bronchitis is a frequent accompaniment, particularly in the winter. Sudden attacks of oppressed breathing, dyspnœa particularly at night, are not infrequent. This is often a uræmic symptom, but is sometimes cardiac. The patient may sit up in bed and gasp for breath, as in true asthma. Cheyne-Stokes breathing may be present, most commonly toward the close, but the patient may be walking about and even attending to his occupation.

Digestive System.—Dyspepsia and loss of appetite are common. Severe and uncontrollable vomiting may be the first symptom. This is usually regarded as a manifestation of uræmia, but it may occur without any other

indications, and I have known it to prove fatal without any suspicion that chronic Bright's disease was present. Severe and even fatal diarrhœa may develop. The tongue may be coated and the breath heavy and urinous.

Nervous System.—Various cerebral manifestations have already been mentioned under uræmia. Headache, sometimes of the migraine type, may be an early and persistent feature of chronic Bright's disease. Cerebral apoplexy is closely related to interstitial nephritis. The hæmorrhage may take place into the meninges or the cerebrum. It is usually associated with marked changes in the vessels. Neuralgias, in various regions, are not uncommon.

Special Senses.—Troubles in vision may be the first symptom of the disease. It is remarkable in how many cases of interstitial nephritis the condition is diagnosed first by the ophthalmic surgeon. The flame shaped retinal hæmorrhages are the most common. Less frequent is diffuse retinitis or papillitis. Sudden blindness may supervene without retinal changes—uræmic amaurosis. Diplopia is a rare event. Recurring conjunctival and palpebral hæmorrhages are fairly common, particularly in the arterio-sclerotic form. Auditory troubles are by no means infrequent in chronic Bright's disease. Ringing in the ears, with dizziness, is not uncommon. Various forms of deafness may occur. Epistaxis is not infrequent, either alone, or of a severe type in association with purpura.

Skin.—Œdema is not common in interstitial nephritis. Slight puffiness of the ankles may be present, but in a majority of the cases dropsy does not supervene. When extensive, it is almost always the result of gradual failure of the hypertrophied heart. The skin is often dry and pale, and sweats are not common. In some instances the sweat may deposit a white frost of urea on the surface of the skin. Eczema is a common accompaniment of chronic interstitial nephritis. Tingling of the fingers or numbness and pallor—the dead fingers—are not, as some suppose, in any way peculiar to Bright's disease. Intolerable itching of the skin may be present, and cramps in the muscles are by no means rare.

Hæmorrhages are not infrequent; epistaxis may prove serious and extensive; purpura may occur. Broncho-pulmonary hæmorrhages are said, by some French writers, to be common, but no instance of it has come under my observation. Ascites is rare except in association with cirrhosis of the liver.

Diagnosis.—The autopsy often discloses the true nature of the disease, one of the many intercurrent affections of which may have proved fatal. The early stages of interstitial nephritis are not recognizable. In a patient with increased pulse tension (particularly if the vessel wall is sclerotic), with the apex beat of the heart dislocated to the left, the second aortic sound ringing and accentuated, the urine abundant and of low specific gravity, with a trace of albumin and an occasional hyaline or granular cast, the diagnosis of interstitial nephritis may be safely made. Of all the indications, that offered by the pulse is the most important. Persistent high tension with thickening of the arterial wall in a man under fifty means that serious mischief has already taken place, that cardio-vascular changes are certainly, and renal most probably, present. In the arterio-sclerotic cases the history is of the "strenuous life"—work, alcohol, tobacco, Venus—and not of an infection or of lead or gout. The urine is not of persistently low specific gravity, there may be little or

Morning headaches in sand-blast cases. age 45-60

headache walker early + lasts little about noon. (posture in bed?)

Brain work upsets/brings on headache / common in hypertension.

Tr. no headache days on tobacco. - milk diet.

Ambard's coeffs expresses the relation between the concentration of urea in blood + rate of excretion in urine.

Functional Tests. urea of non-protein + urea N. of blood must be studied in relation one to retention alone or retention + protein declination - former better diagnosis.

Ambard's coeff. [illegible] degree ability of kidney to excrete urea than amount of urea in blood

Prognosis - best followed by phthalein test + Ambard's coeffs

normal test kills everything.

increase of uric acid in blood - value as early sign of nephritis

urea in blood - useful as a guide to fr. of nitrogenous

Impregnemia - blood creatinin retained - over 5 mg. per 100 cc
fatal in short time. Interstitial stage 15 to 30 mg per cent

In diabetes + nephritis. ant. of sugar in absence little value as
nephritis has limited permeability of kid. for sugar

Estim. of sugar in blood -

no albumin except in intercurrent attacks; the symptoms are cardiac rather than renal or cerebral; the ocular changes are hæmorrhagic, not the true albuminuric retinitis

Prognosis.—Chronic Bright's disease is an incurable affection, and the anatomical conditions on which it depends are quite as much beyond the reach of medicines as wrinkled skin or gray hair. Interstitial nephritis, however, is compatible with the enjoyment of life for many years, and it is now universally recognized that increased tension, thickening of the arterial walls, and polyuria with a small quantity of albumin, neither doom a man to death within a short time nor necessarily interfere with the pursuits of an active life so long as proper care be taken. I know patients who have had high tension and a little albumin in the urine with hyaline casts for ten, twelve, or even fifteen years. Serious indications are the occurrence of uræmic symptoms, dilatation of the heart, the onset of serous effusions, the onset of Cheyne-Stokes breathing, persistent vomiting, and diarrhœa. The phenolsulphonephthalein test gives valuable information as to the functional capacity of the kidneys and is a material aid in prognosis.

Treatment.—Patients without local indications or in whom the condition has been accidentally discovered should so regulate their lives as to throw the least possible strain upon heart, arteries, and kidneys. A quiet life without mental worry, with gentle but not excessive exercise, and residence in an equable climate, should be recommended. In addition they should be told to keep the bowels regular, the skin active by a daily tepid bath with friction, and the urinary secretion free by drinking daily a definite amount of either distilled water or some pleasant mineral water. Alcohol should be strictly prohibited. Tea and coffee are allowable.

The diet should be light and nourishing, and the patient should be warned not to eat excessively, and not to take meat more than once a day. Care in food and drink is probably the most important element in the treatment of these early cases.

A patient in good circumstances may be urged to go away during the winter months, or, if necessary, to move altogether to a warm equable climate, like that of Southern California. There is no doubt of the value in these cases of removal from the changeable, irregular weather which prevails in the temperate regions from November until April.

At this period medicines are not required unless for certain special symptoms. Patients derive much benefit from an annual visit to certain mineral springs, such as Poland, Bedford, Saratoga, in America, and Vichy and others in Europe. Mineral waters have no curative influence upon chronic Bright's disease; they simply help the interstitial circulation and keep the drains flushed. In this early stage, when the patient's condition is good, the tension not high, and the quantity of albumin small, medicines are not indicated, since no remedies are known to have the slightest influence upon the progress of the disease. Sooner or later SYMPTOMS arise which demand treatment. Of these the following are the most important:

(*a*) *Greatly Increased Arterial Tension.*—It is to be remembered that a certain increase of tension is not only necessary but unavoidable in chronic Bright's disease, and probably the most serious danger is too great lowering of the blood tension. The happy medium must be sought between such

heightened tension as throws a serious strain upon the heart and risks rupture of the vessels and the low tension which, under these circumstances, is specially liable to be associated with serous effusions. In cases with persistent high tension the diet should be light, an occasional saline purge should be given, and sweating promoted by means of hot air or the hot bath. If these measures do not suffice, nitroglycerin may be tried, beginning with 1 minim of the 1-per-cent. solution three times a day, and gradually increasing the dose if necessary. Patients vary so much in susceptibility to this drug that in each case it must be tested, the limit of dosage being that at which the patient experiences the physiological effect. As much as 10 minims of the 1-per-cent. solution may be given three times a day. In many cases I have given it in much larger doses for weeks at a time. I have never seen any ill effects from it. If the dose is excessive the patients complain at once of flushing or headache. Its use may be kept up for six or seven weeks, then stopped for a week and resumed. Its value is seen not only in the reduction of the tension, but also in the striking manner in which it relieves the headache, dizziness, and dyspnœa. The sodium nitrite may be given in doses of grs. ii-v (0.13 to 0.3 gm.) three times a day.

(*b*) More or less *anæmia* is present in advanced cases, and is best met by the use of iron. Weir Mitchell, who has had a unique experience in certain forms of chronic Bright's disease, gives the tincture of the perchloride of iron in large doses—from half a drachm to a drachm three times a day. He thinks that it not only benefits the anæmia, but that it also is an important means of reducing the arterial tension.

(*c*) Many patients with Bright's disease present themselves for treatment with signs of cardiac dilatation; there is a gallop rhythm or the heart-sounds have a fetal character, the breath is short, the urine scanty and highly albuminous, and there are signs of local dropsy. In these cases the treatment must be directed to the heart. A morning dose of salts or calomel may be given, and digitalis in 10-minim doses, three or four times a day. Strychnia may be used with benefit in this condition. In some instances other cardiac tonics may be necessary, but as a rule the digitalis acts promptly and well.

(*d*) *Uræmic Symptoms.*—Even before marked manifestations are present there may be extreme restlessness, mental wandering, a heavy, foul breath, and a coated tongue. Headache is not often complained of, though intense frontal headache may be an early symptom of uræmia. In this condition, too, the patient may complain of palpitation, feelings of numbness, and sometimes nocturnal cramps. For these symptoms the saline purgatives should be ordered, and hot baths, so as to induce copious sweating. Grandin states that irrigation of the bowel with water at a temperature from 120° to 150° is most useful. Nitroglycerin also may be used to reduce the tension. For the uræmic convulsions, if severe, inhalations of chloroform may be used. If the patient is robust and full-blooded, from 12 to 20 ounces of blood should be removed. The patient should be freely sweated, and if the convulsions tend to recur chloral may be given, either by the mouth or per rectum, or, better still, morphia. Uræmic coma must be treated by active purgation, and sweating should be promoted by the use of pilocarpine or the hot bath. For the restlessness and delirium morphia is indispensable. Since its recommendation in uræmic states some years ago, by Stephen MacKenzie, I have

used this remedy extensively and can speak of its great value in these cases. I have never seen ill effects or any tendency to coma follow. It is of special value in the dyspnœa and Cheyne-Stokes breathing of advanced arterio-sclerosis with chronic uræmia.

Surgical Treatment.—Edebohls introduced the operation of decapsultation of the kidneys ~~in Bright's disease~~ in order to establish new vascular connections, and so influence the nutrition and work of the organs. ~~In~~ his work records are given of 72 cases; 7 died within two weeks, 22 died at periods more or less remote, 3 disappeared from observation, and 40 were known to be living—one eleven years and eight months after the operation. As Edebohls said the difficult thing to determine is the existence of chronic Bright's disease before operation. No case should be regarded as such on the urine examination alone. The cardio-vascular condition and the retinæ should be studied. There is probably a small group of suitable cases—the subacute and chronic forms which follow the acute infections—in which the outlook is hopeless from medical treatment.

VIII. AMYLOID DISEASE

Amyloid (lardaceous or waxy) degeneration of the kidneys is simply an event in the process of chronic ~~Bright's disease~~, most commonly in the chronic parenchymatous nephritis following fevers, or of cachectic states. It has no claim to be regarded as one of the varieties of ~~Bright's disease~~. The affection of the kidneys is generally a part of a widespread amyloid degeneration occurring in prolonged suppuration, as in disease of the bone, in syphilis, tuberculosis, and occasionally leukæmia, lead poisoning, and gout. It varies curiously in frequency in different localities.

Anatomically the amyloid kidney is large and pale, the surface smooth, and the venæ stellatæ well marked. On section the cortex is large and may show a peculiar glistening, infiltrated appearance, and the glomeruli are very distinct. The pyramids, in striking contrast to the cortex, are of a deep red color. A section soaked in dilute tincture of iodine shows spots of a walnut or mahogany brown color. The Malpighian tufts and the straight vessels may be most affected. In lardaceous disease of the kidneys the organs are not always enlarged. They may be normal in size or small, pale, and granular. The amyloid change is first seen in the Malpighian tufts, and then involves the afferent and efferent vessels and the straight vessels. It may be confined entirely to them. In later stages of the disease the tubules are affected, chiefly the membrane, rarely, if ever, the cells themselves.

Symptoms.—The renal features alone may not indicate the presence of this degeneration. Usually the associated condition gives a hint of the nature of the process. The urine, as a rule, shows important changes; the quantity is increased, and it is pale, clear, and of low specific gravity. The albumin is usually abundant, but it may be scanty, and in rare instances absent. Possibly the variations in the situation of the amyloid changes may account for this, since albumin is less likely to be present when the change is confined to the vasa recta. In addition to ordinary albumin globulin may be present. The tube casts are variable, usually hyaline, often fatty or finely granular. Occasionally the amyloid reaction can be detected in the hyaline casts. Dropsy

is present in many instances, particularly when there is much anæmia or profound cachexia. It is not, however, an invariable symptom, and there are cases in which it does not develop. Diarrhœa is a common accompaniment.

Increased arterial tension and cardiac hypertrophy are not usually present, except in those cases in which amyloid degeneration occurs in the secondary contracted kidney; under which circumstances there may be uræmia and retinal changes, which, as a rule, are not met with in other forms.

Diagnosis.—By the condition of the urine alone it is not possible to recognize amyloid changes in the kidney. Usually, however, there is no difficulty, since the ~~Bright's disease~~ comes on in association with syphilis, prolonged suppuration, disease of the bone, or tuberculosis, and there is evidence of enlargement of the liver and spleen. A suspicious circumstance is the existence of polyuria with a large amount of albumin in the urine and few casts, or when, in these constitutional affections, a large quantity of clear, pale urine is passed, even without the presence of albumin.

The prognosis depends rather on the condition with which the nephritis is associated. As a rule it is grave.

IX. PYELITIS

(*Consecutive Nephritis; Pyelonephritis; Pyonephrosis*)

Definition.—Inflammation of the pelvis of the kidney and the conditions which result from it.

Etiology.—Pyelitis in almost all cases is induced by bacterial invasion and multiplication, rarely by the irritation of various substances such as turpentine, cubebs, or sugar (diabetes). Normally the kidney can eliminate without harm to itself, apparently, various bacteria carried to it by the blood-current from the intestinal tract or some focus of infection; and it probably becomes infected only when its resistance is lowered, as a result of some general cause, as anæmia, malnutrition, or intercurrent disease, or of some local cause, as nephritis, displacement, congestion due to pressure of neoplasms upon the ureter, twisted ureter (Dietl's crisis), or of operation, or when the number or virulence of the micro-organisms is increased. These same factors probably play an important rôle also in the other common causes of pyelitis, ascending infection from an infected bladder (cystitis), and tuberculous infection. Other causes described are various fevers, cancer, hydatids, the ova of certain parasites, cold, and overexertion. Calculus seems not to be a common cause. It is a not uncommon complication of pregnancy (French). In T. R. Brown's series of 20 cases the colon bacillus was obtained 7 times, the tubercle bacillus 6, the proteus bacillus 4, a white staphylococcus twice, while in 1 case cultures were negative.

Morbid Anatomy.—In the early stages of pyelitis the mucous membrane is turbid, somewhat swollen, and may show ecchymoses or a grayish pseudomembrane. The urine in the pelvis is cloudy, and, on examination, numbers of epithelial cells are seen.

In the calculous pyelitis there may be only slight turbidity of the membrane, which has been called by some catarrhal pyelitis. More commonly the

mucosa is roughened, grayish in color, and thick. Under these circumstances there is almost always more or less dilatation of the calyces and flattening of the papillæ. Following this condition there may be (*a*) extension of the suppurative process to the kidney itself, forming a pyelonephritis; (*b*) a gradual dilatation of the calyces with atrophy of the kidney substance, and finally the production of the condition of pyonephrosis, in which the entire organ is represented by a sac of pus with or without a thin shell of renal tissue. (*c*) After the kidney structure has been destroyed by suppuration, if the obstruction at the orifice of the pelvis persists, the fluid portions may be absorbed and the pus become inspissated, so that the organ is represented by a series of sacculi containing grayish, putty like masses, which may become impregnated with lime salts.

Tuberculous pyelitis, as already described, usually starts upon the apices of the pyramids, and may at first be limited in extent. Ultimately the condition produced may be similar to that of calculous pyelitis. Pyonephrosis is quite as frequent a sequence, while the final transformation of the pus into a putty-like material impregnated with salts, forming the so-called scrofulous kidney, is even commoner.

The pyelitis consecutive to cystitis is generally bilateral, and the kidneys are sometimes involved, forming the so-called "*surgical kidneys*"—acute suppurative nephritis. There are lines of suppuration extending along the pyramids, or small abscesses in the cortex, often just beneath the capsule; or there may be wedge shaped abscesses. The pus organisms either pass up the tubules or, as Steven has shown, through the lymphatics.

Symptoms.—The forms associated with the fevers rarely cause any symptoms, even when the process is extensive. In mild grades there is pain in the back or there may be tenderness on deep pressure on the affected side. The urine, turbid and containing pus cells, some mucus, and occasional red blood-cells, is acid or alkaline, depending on the infecting microbe; usually the albuminuria is of higher grade comparatively than the pyuria.

Before the condition of pyuria is established there may be attacks of pain on the affected side (not reaching the severe agony of renal colic), rigors, high fever, and sweats. Under these circumstances the urine, which may have been clear, becomes turbid or smoky from the presence of blood, and may contain large numbers of mucus cells and transitional epithelium.

The statement is not infrequently made that the epithelium in the urine in pyelitis is distinctive and characteristic. This is erroneous, as may be readily demonstrated by comparing scrapings of the mucosa of the renal pelvis and of the bladder. In both the epithelium belongs to what is called the transitional variety, and in both regions the same conical, fusiform, and irregular cells with long tails are found, and yet in pyelitis more of these tailed cells occur, for in cystitis one must often search long for them.

When the pyelitis, whether calculous or tuberculous, has become chronic and discharges, the symptoms are:

(*a*) *Pyuria.*—The pus is in variable amount, and may be intermittent. Thus, as is often the case when only one kidney is involved, the ureter may be temporarily blocked, and normal urine is passed for a time; then there is a sudden outflow of the pent up pus and the urine becomes purulent. Coincident with this retention, a tumor mass may be felt on the side affected. The

pus has the ordinary characters, but the transitional epithelium is not so abundant at this stage and comes from the bladder or from the pelvis of the healthy side. Occasionally, in rapidly advancing pyelonephritis, portions of the kidney tissue, particularly of the apices of the pyramids, may slough away and appear in the urine; or, as in a remarkable specimen shown to me by Tyson, solid cheesy moulds of the calyces are passed. Casts from the kidney tubules are sometimes present. The reaction of the urine depends entirely upon the infecting microbe, whether the condition is unilateral or bilateral, and whether the bladder is also infected, when vesical irritability and frequent micturition may be present. Polyuria is usually present in the chronic cases.

(*b*) Intermittent fever associated with rigors is usually present in cases of suppurative pyelitis. The chills may recur at regular intervals, and the cases are often mistaken for malaria. Owen-Rees called attention to the frequent occurrence of these rigors, which form a characteristic feature of both calculous and tuberculous pyelitis. Ultimately the fever assumes a hectic type and the rigors may cease.

(*c*) The general condition of the patient often indicates prolonged suppuration. There is more or less wasting with anæmia and a progressive failure of health. Secondary abscesses may develop and the clinical picture becomes that of pyæmia. In some instances, particularly of tuberculous pyelitis, the clinical course may resemble that of typhoid fever. There are instances of pyuria recurring, at intervals, for many years without impairment of the bodily vigor. Some of the chronic cases have practically no discomfort.

(*d*) Physical examination in chronic pyelitis usually reveals tenderness on the affected side or a definite swelling, which may vary much in size and ultimately attain large dimensions if the kidney becomes enormously distended, as in pyonephrosis.

(*e*) Occasionally nervous symptoms, which may be associated with dyspnœa, supervene, or the termination may be by coma, not unlike that of diabetes. These have been attributed to the absorption of the decomposing materials in the urine, whence the so-called ammoniæmia. A form of paraplegia has been described in connection with some cases of abscess of the kidney, but whether due to a myelitis or to a peripheral neuritis has not yet been determined.

In suppurative nephritis or surgical kidney following cystitis, the patient complains of pain in the back, the fever becomes high, irregular, and associated with chills, and in acute cases a typhoid state may precede the fatal event.

Diagnosis.—Between the tuberculous and the calculous forms of pyelitis it may be difficult or impossible to distinguish, except by the detection of tubercle bacilli in the pus. The examination for bacilli should be made systematically, and in suspicious cases intraperitoneal injections of guinea-pigs should also be made. From perinephric abscess pyonephrosis is distinguished by the more definite character of the tumor, the absence of œdematous swelling in the lumbar region, and, most important of all, the history of the case. The urine, too, in perinephric abscess may be free from pus. There are cases, however, in which it is difficult indeed to make a satisfactory diagnosis.

Suppurative pyelitis and cystitis are apt to be confounded, and perineal

In diag. from pyelitis from pyelo-nephritis functional test is important + functional test reduced in pyelo-neph. without pyelitis

section is not infrequently performed on the supposition of the existence of the latter. The two conditions may, of course, coexist and prove puzzling, but the history, the higher relative grade of albuminuria in pyelitis, the polyuria, the mode of development, the local signs in one lumbar region, and the absence of pain in the bladder should be sufficient to differentiate the affections. In women by catheterization of the ureters, it may be definitely determined whether the pus comes from the kidneys or from the bladder. The cystoscope may be used for this purpose.

Much may be done with X-ray examinations to determine the condition of the pelves of the kidneys. When a 2-per-cent. solution of collargol is injected by means of the ureteral catheter a shadow is cast giving a very accurate outline of the pelvis of the organ.

Prognosis.—Cases coming on during the fevers usually recover. Tuberculous pyelitis may terminate favorably by inspissation of the pus and conversion into a putty-like substance with deposition of lime salts. With pyonephrosis the dangers are increased. Perforation may occur into the peritoneum, the patient may be worn out by the hectic fever, or amyloid disease may develop.

Treatment.—Fluids should be taken freely, particularly the alkaline mineral waters, to which potassium citrate may be added.

The treatment of the calculous form will be considered later. Practically there are no remedies which have much influence upon the pyuria. Some of the urinary antiseptics seem to be of value, especially in the acute cases. Urotropin should be given in full doses (gr. xv, 1 gm., three or four times a day); watch should be kept for signs of irritation and the dose reduced if they appear. Vaccine therapy is sometimes of value. Tonics should be given, a nourishing diet, and milk and butter-milk may be taken freely. When the tumor has formed or even before it is perceptible, if the symptoms are serious and severe, the kidney should be explored, and, if necessary, nephrotomy or nephrectomy should be performed.

X. HYDRONEPHROSIS

Definition.—Dilatation of the pelvis and calyx of the kidney with atrophy of its substance, caused by the accumulation of non-purulent fluids, the result of obstruction.

Etiology.—The condition may be congenital, owing to some abnormality in the ureter or urethra. The tumor produced may be large enough to retard labor. Sometimes it is associated with other malformations. There is a condition of moderate dilatation, apparently congenital, which is not connected with any obstruction in the ducts.

In some instances there has been contraction or twisting of the ureter, or it has been inserted into the kidney at an acute angle or at a high level. In adult life the condition may be due to lodgment of a calculus, or to a cicatricial stricture following ulcer.

There is a remarkable condition of hypertrophy and dilatation of the bladder and ureters associated with congenital defect of the abdominal muscles. The bladder may form a large abdominal tumor and the ureters may be as large and visible as coils of the small intestine.

New growths, such as tubercle or cancer, occasionally induce hydronephrosis; more commonly, pressure upon the ureter from without, particularly tumors of the ovaries and uterus. Occasionally cicatricial bands compress the ureter. Obstruction within the bladder may result from cancer, from hypertrophy of the prostate with cystitis, and in the urethra from stricture. It is stated that slight grades of hydronephrosis have been found in patients with excessive polyuria.

In whatever way produced, when the ureter is blocked the secretion accumulates in the pelvis and infundibula. Sometimes acute inflammation follows, but more commonly the slow, gradual pressure causes atrophy of the papillæ with gradual distention and wasting of the organ. In acquired cases from pressure, even when dilatation is extreme, there may usually be seen a thin layer of renal structure. In the most extreme stages the kidney is represented by a large cyst, which may perhaps show on its inner surface imperfect septa. The fluid is thin and yellowish in color, and contains traces of urinary salts, urea, uric acid, and sometimes albumin. The secretion may be turbid from admixture with small quantities of pus.

Total occlusion does not always lead to a hydronephrosis, but may be followed by atrophy of the kidney. It appears that when the obstruction is intermittent or not complete the greatest dilatation is apt to follow. The sac may be enormous, and cause an abdominal tumor of the largest size. The condition has even been mistaken for ascites. Enlargement of the other kidney may compensate for the defect. Hypertrophy of the left side of the heart usually follows.

Symptoms.—When small, it may not be noticed. The congenital cases when bilateral usually prove fatal within a few days; when unilateral, the tumor may not be noticed for some time. It increases progressively and has all the characters of a tumor in the renal region. In adult life many of the cases, due to pressure by tumors, as in cancer of the uterus and enlargement of the prostate, etc., give rise to no symptoms.

In *intermittent* hydronephrosis the tumor suddenly disappears with the discharge of a large quantity of clear fluid; the sac gradually refills, and the process may be repeated for years. In these cases the obstruction is unilateral; a cicatricial stricture exists, or a valve is present in the ureter, or the ureter enters the upper part of the pelvis. Many of the cases are in women and associated with movable kidney.

The examination of the abdomen shows, in unilateral hydronephrosis, a tumor occupying the renal region. When of moderate size it is readily recognized, but when large it may be confounded with ovarian or other tumors. In young children it may be mistaken for sarcoma of the kidney or of the retroperitoneal glands, the common cause of abdominal tumor in early life. Aspiration alone would enable us to differentiate between hydronephrosis and tumor. The large hydronephrotic sac is frequently mistaken for ovarian tumor. The latter is, as a rule, more mobile, and rarely fills the deeper portion of the lumbar region so thoroughly. The ascending colon can often be detected passing over the renal tumor, and examination per vaginam, particularly under ether, will give important indications as to the condition of the ovaries. In doubtful cases the sac should be aspirated. The fluid of the renal cyst is clear, or turbid from the presence of cell elements, rarely colloid in

character; the specific gravity is low; albumin and traces of urea and uric acid are usually present; and the epithelial elements in it may be similar to those found in the pelvis of the kidney. In old sacs, however, the fluid may not be characteristic, since the urinary salts disappear, but in one case of several years' duration oxalates of lime and urea were found.

Perhaps the greatest difficulty is offered by the condition of hydronephrosis in a movable kidney. Here, the history of sudden disappearance of the tumor with the passage of a large quantity of clear fluid would be a point of great importance in the diagnosis. In those rare instances of an enormous sac filling the entire abdomen, and sometimes mistaken for ascites, the character of the fluid might be the only point of difference. The tumor of pyonephrosis may be practically the same in physical characteristics. Fever is usually present, and pus is often found in the urine. In these cases, when in doubt, exploratory puncture should be made.

The outlook in hydronephrosis depends much upon the cause. When single, the condition may never produce serious trouble, and the intermittent cases may persist for years, and finally disappear. Occasionally the cyst ruptures into the peritoneum, more rarely through the diaphragm into the lung. A remarkable case of this kind was under the care of my colleague, Halsted. A man, aged twenty-one, had, from his second year, attacks of abdominal pain in which a swelling would appear between the hip and costal margin and subside with the passage of a large amount of urine. In January, 1888, the sac discharged through the right lung. Reaccumulations occurred on several occasions, and on June 9, 1891, the sac was opened and drained. He remains well, though there is still a sinus through which a clear, probably urinous, fluid is discharged.

The sac may discharge spontaneously through the ureter and the fluid never reaccumulate. In bilateral hydronephrosis there is a danger that uræmia may supervene. There are instances, too, in which blocking of the ureter on the sound side by calculus has been followed by uræmia. And, lastly, the sac may suppurate, and the condition change to one of pyonephrosis.

Treatment.—Cases of intermittent hydronephrosis which do not cause serious symptoms should be let alone. It is stated that, in sacs of moderate size, the obstruction has been overcome by shampooing. If practiced, it should be done with great care. When the sac reaches a large size aspiration may be performed and repeated if necessary. Puncture should be made in the flank, midway between the ilium and the last rib. If the fluid reaccumulates and the sac becomes large, it may be incised and drained, or, as a last resort, the kidney may be removed. In women a carefully adapted pad and bandage will sometimes prevent the recurrence of an intermittent hydronephrosis.

XI. NEPHROLITHIASIS

(*Renal Calculus*)

Definition.—The formation in the kidney or in its pelvis of concretions, by the deposition of certain of the solid constituents of the urine.

Etiology and Pathology.—In the kidney substance itself the separation of the urinary salts produces a condition to which, unfortunately, the term

infarct has been applied. Three varieties may be recognized: (1) The uric acid infarct, usually met with at the apices of the pyramids in new born children and during the first weeks of life. The priapism and attacks of crying in the new-born have been attributed to the passage of these infarcts; (2) the sodium urate infarct, sometimes associated with ammonium urate, which forms whitish lines at the apices of the pyramids and is met with chiefly, but not always, in gouty persons; and (3) the lime infarcts, forming very opaque white lines in the pyramids, usually in old people.

In the pelvis and calyces concretions of the following forms occur: (*a*) Small gritty particles, *renal sand*, ranging in size from the individual grains of the uric acid sediment to bodies 1 or 2 mm. in diameter. These may be passed in the urine for long periods without producing any symptoms, since they are too fine to be arrested in their downward passage.

(*b*) Larger concretions, ranging in size from a small pea to a bean, and either solitary or multiple in the calyces and pelvis. It is the smaller of these calculi which, in their passage, produce the attacks of renal colic. They may be rounded and smooth, or present numerous irregular projections.

(*c*) The dendritic form of calculus. The orifice of the ureter may be blocked by a Y-shaped stone. The pelvis itself may be occupied by the concretion, which forms a more or less distinct mould. These are the remarkable *coral calculi*, which form in the pelvis complete moulds of infundibula and calyces, the latter even presenting cup-like depressions corresponding to the apices of the papillæ. Some of these casts in stone of the renal pelvis are as beautifully moulded as Hyrtl's corrosion preparations.

Chemically the varieties of calculi are: (1) Uric acid and urates, most important, and forming the renal sand, the small solitary, or the large dendritic stones. They are very hard, the surface is smooth, and the color reddish. The larger stones are usually stratified and very dense. Usually the uric acid and the urates are mixed, but in children stones composed of urates alone may occur.

(2) Oxalate of lime, which forms mulberry-shaped calculi, studded with points and spines. They are often very dark in color, intensely hard, and are a mixture of oxalate of lime and uric acid.

(3) Phosphatic calculi are composed of the calcium phosphate and the ammonio-magnesium phosphate, sometimes mixed with a small amount of calcium carbonate. They are quite common, although the phosphatic salts are often deposited about the uric acid or the calcium oxalate stones.

(4) Rare forms of calculi are made up of cystine, xanthine, carbonate of lime, indigo, and urostealith.

The mode of formation of calculi has been much discussed. They may be produced by an excess of a sparingly soluble abnormal ingredient, such as cystine or xanthine; more frequently by the presence of uric acid in a very acid urine which favors its deposition. Sir William Roberts thus briefly states the conditions which lead to the formation of the uric acid concretions: high acidity, poverty in salines, low pigmentation, and high percentage of uric acid. Ord suggests that albumin, mucus, blood, and epithelial threads may be the starting point of stone. The demonstration of organisms in the centre of renal calculi renders it probable that in many cases the nucleus of the stone is an agglutinated mass of bacteria.

chem. compos. Rosenheim. J. a. M. a. July 10. 15

Majority of Calcium Oxalate + not of uric acid crystals

1926 - salts of uric acid. No mostly of Calcium salts.

Renal calculi are most common in the early and later periods of life. They are moderately frequent in the United States, but there do not appear to be special districts, corresponding to the "stone counties" in England. Men are more often affected than women. Sedentary occupations seem to predispose to stone.

The effects of the calculi are varied. It is by no means uncommon to find a dozen or more stones of various sizes in the calyces without any destruction of the mucous membrane or dilatation of the pelvis. A turbid urine fills the pelvis, in which there are numerous cells from the epithelial lining. There are cases of this sort in which, apparently, the stones may go on forming and are passed for years without seriously impairing the health and without inconvenience, except the attacks of renal colic. Still more remarkable are the cases of coral like calculi, which may occupy the entire pelvis and calyces without causing pyelitis, but which gradually lead to more or less induration of the kidney. The most serious effects are when the stone excites a suppurative pyelitis and pyonephrosis.

Symptoms.—Patients may pass gravel for years without having an attack of renal colic, and a stone may never lodge in the ureter. In other instances, the formation of calculi goes on year by year and the patient has recurring attacks such as have been so graphically described by Montaigne in his own case. A patient may pass enormous numbers of calculi. A patient may pass a single calculus, and never be troubled again. The large coral calculi may excite no symptoms. In a remarkable specimen of the kind, presented to the McGill Medical Museum by J. A. Macdonald, the patient, a middle-aged woman, died suddenly with uræmic symptoms. There was no pyelitis, but the kidneys were sclerotic.

Renal colic ensues when a stone enters the ureter, or follows an acute pyelitis. An attack may set in abruptly without apparent cause, or may follow a strain in lifting. It is characterized by agonizing pain, which starts in the flank of the affected side, passes down the ureter, and is felt in the testicle and along the inner side of the thigh. The pain may also radiate through the abdomen and chest, and be very intense in the back. In severe attacks there are nausea and vomiting and the patient is collapsed. The perspiration breaks out upon the face and the pulse is feeble and quick. A chill may precede the outbreak, and the temperature may rise as high as 103°. No one has more graphically described an attack of "the stone" than Montaigne,* who was a sufferer for many years: "Thou art seen to sweat with pain, to look pale and red, to tremble, to vomit well-nigh to blood, to suffer strange contortions and convulsions, by starts to let tears drop from thine eyes, to urine thick, black, and frightful water, or to have it suppressed by some sharp and craggy stone, that cruelly pricks and tears thee." From personal experience, I can describe three sorts of pain in an attack of renal colic: (*a*) A constant localized, dull pain, the area of which could be covered on the skin of the back in the renal region by a penny piece, and which could be imitated exactly by deep firm pressure on a superficial bone. (*b*) Paroxysms of pain radiating in the course of the ureter or into the flank, and as they increase accompanied by sweating, fainting, and nausea. (*c*) Flushes or

*Essays, Book 111, 13.

rushes of hot pain at intervals, often momentary, usually passing to the back, less often toward the groin. Dozens of these flushes relieved the monotony of (*b*). The symptoms persist for a variable period. In short attacks they do not last longer than an hour; in other instances they continue for a day or more, with temporary relief. Micturition is frequent, occasionally painful, and the urine, as a rule, is bloody. There are instances in which a large amount of clear urine is passed, probably from the other kidney. In rare cases the secretion of urine is completely suppressed, even when the kidney on the opposite side is normal, and death may occur from uræmia. This most frequently happens when the second kidney is extensively diseased, or when only a single kidney exists.

After the attack of colic has passed there is more or less aching on the affected side, and the patient can usually tell from which kidney the stone has come. Examination during the attack is usually negative. Very rarely is the kidney ~~becomes~~ palpable. Tenderness on the affected side is common. In very thin persons it may be possible, on examination of the abdomen, to feel the stone in the ureter; or the patient may complain of a grating sensation.

When the calculi remain in the kidney they may produce very definite and characteristic symptoms, of which the following are the most important:

(*a*) *Pain,* usually in the back, which is often no more than a dull soreness, but which may be severe and come on in paroxysms. It is usually on the side affected, but may be referred to the opposite kidney, and there are instances in which the pain has been confined to the sound side. It radiates in the direction of the ureter, and may be felt in the scrotum or even in the penis. Pains of a similar nature may occur in movable kidneys, ~~and there are several instances on record in which~~ surgeons have incised the kidney for stone and found none. In an instance in which pain was present for a couple of years the exploration revealed only a contracted kidney.

(*b*) *Hæmaturia.*—Although this occurs most frequently when the stone becomes engaged in the ureter, it may also come on when the stones are in the pelvis. The bleeding is seldom profuse, as in cancer, but in some instances may persist for a long time. It is aggravated by exertion and lessened by rest. Frequently it only gives to the urine a smoky hue. The urine may be free for days, and then a sudden exertion or a prolonged ride may cause smokiness, or blood may be passed in considerable quantities.

(*c*) *Pyelitis.*—(1) There may be attacks of severe pain in the back, not amounting to actual colic, which are initiated by a heavy chill followed by fever, in which the temperature may reach 104° or 105°, followed by profuse sweating. The urine, which has been clear, may become turbid and smoky and contain blood and abundant epithelium from the pelvis. Attacks of this description may recur at intervals for months or even years, and are generally mistaken for malaria, unless special attention is paid to the urine and to the existence of the pain in the back. This renal intermittent fever, due to the presence of calculi, is analogous to the hepatic intermittent fever, due to gall-stones, and in both it is important to remember that the most intense paroxysms may occur without any evidence of suppuration.

(2) More frequently the symptoms of purulent pyelitis, which have already been described, are present: pain in the renal region, recurring chills, and pus in the urine, with or without indications of pyonephrosis.

Vanilla [illegible]

Orchilis [illegible]

or sic prostatic' dio carel

Medical institutions

→ Semi-medicines and with practical diseases

Tr. uric acid stones – add. of alkalies

Cal. ox. + phos. – deposited in alkaline medicines + dissolved by acids

advantage if any stone could be analyzed

(*d*) *Pyuria.*—There are instances of stone in the kidney in which pus occurs continuously or intermittently in the urine for many years.

Patients with stone in the kidney are often robust, high livers, and gouty. Attacks of dyspepsia are not uncommon, or they may have severe headaches.

Diagnosis.—The X-ray picture is rarely at fault, and specialists in this department are becoming more and more skillful, so that mistakes are now rare. Renal may be mistaken for intestinal colic, particularly if the distention of the bowels is marked, or for biliary colic. The situation and direction of the pain, the retraction and tenderness of the testicle, the occurrence of hæmaturia, and the altered character of the urine are distinctive features. Attention may again be called to the fact that attacks simulating renal colic are associated with movable kidney, or even, it has been supposed, without mobility of the kidney, with the accumulation of the oxalates or uric acid in the pelvis of the kidney. The diagnosis between a stone in the kidney and stone in the bladder is not always easy, though in the latter the pain is particularly about the neck of the bladder, and not limited to one side. In the uric acid or uratic renal stone, the urine is acid, thus aiding us in differentiating it from a bladder stone, when alkaline urine is the rule. It is stated that certain differences occur in the symptoms produced by different sorts of calculi. The large uric acid calculi less frequently produce severe symptoms. On the other hand, as the oxalate of lime is a rougher calculus, it is apt to produce more pain (often of a radiating character) than the lithic acid form, and to cause hæmorrhage. In both these forms the urine is acid. The phosphatic calculi are stated to produce the most intense pain, and the urine is commonly alkaline.

Treatment.—In the attacks of renal colic great relief is experienced by the hot bath, which is sometimes sufficient to relax the spasm. When the pain is very intense morphia should be given hypodermically and inhalations of chloroform may be necessary until the effects of the anodyne are manifest. Local applications are sometimes grateful—hot poultices, or cloths wrung out of hot water. The patient may drink freely of hot lemonade, soda water, or barley water. Occasionally change in posture or inversion will give great relief. Surgical interference should be considered in all cases, especially when the stone is large or the associated pyelitis severe.

In the intervals the patient should, as far as possible, live a quiet life, avoiding sudden exertion of all sorts. The essential feature in the treatment is to keep the urine abundant and, in the uric acid or uratic cases, alkaline. The patient should drink daily a large but definite quantity of mineral waters* or distilled water, which is just as satisfactory. The citrate or bicarbonate of potash may be added. The aching pains in the back are often greatly relieved by this treatment. Many patients find benefit from a stay at Saratoga, Bedford, Poland, or other mineral springs in the United States, or at Vichy or Ems in Europe.

The diet should be carefully regulated, and similar to that indicated in the early stages of gout. Sir William Roberts recommends what is known as the solvent treatment for uric acid calculi. The citrate of potash is given in large doses, half a drachm to a drachm, every three hours in a tumblerful of

*Some of these, if we judge by the laudatory reports, are as potent as the waters of Corsena, declared by Montaigne to be "powerful enough to break stones."

water. This should be kept up for several months. I have had no success with this treatment, nor, when one considers the character of the uric acid stones usually met with in the kidney, does it seem likely that any solvent action could be exercised upon them by changes in the urine. This treatment should be abandoned if the urine becomes ammoniacal.

The value of piperazine as a solvent of uric acid gravel or of uric acid stones has been much discussed. While outside the body a watery solution of the drug has this power in a marked degree, the amount excreted in the urine as given in the ordinary doses of 15 grains daily seems to have very little influence. Several observers have shown that the percentage of piperazine excreted in the urine, when taken in doses of from 1 to 2 grams, has, when tested outside of the body, little or no influence as a solvent (Fawcett, Gordon).

XII. TUMORS OF THE KIDNEY

These are benign and malignant. Of the benign tumors, the most common are the small nodular *fibromata* which occur frequently in the pyramids, and occasionally *lipoma, angioma,* or *lymphadenoma.* The *adenomata* may be congenital. In one of my cases the kidneys were greatly enlarged, contained small cysts, and numerous adenomatous structures throughout both organs.

Malignant growths—*cancer* or *sarcoma*—may be either primary or secondary. The sarcomata are the most common, either alveolar sarcoma or the remarkable form containing striped muscular fibres—rhabdomyoma. One of the most common and important renal tumors is the *hypernephroma,* growing in or upon the organ from the adrenal tissue—the aberrant "rests" of Grawitz. Of 163 cases only 6 were extra-renal (Ellis). They may be small and in the renal cortex or form large tumors with extensive metastases, particularly in the lungs. Most of the primary carcinomas and alveolar sarcomas of the kidney are really hypernephromata. Adami holds that they may arise from either renal or adrenal tissues.

The tumors attain a very large size, and almost fill the abdomen. In children they may be enormous. They grow rapidly, are often soft, and hæmorrhage frequently takes place into them. In the sarcomata, invasion of the pelvis or of the renal vein is common. The rhabdomyomata rarely form very large tumors, and death occurs shortly after birth. In one of my cases the child at the age of three years and a half died suddenly of embolism of the pulmonary artery and tricuspid orifice by a fragment of the tumor, which had grown into the renal vein.

Symptoms.—The following are the most important: (*a*) Hæmaturia in one-half the cases, which may be the first indication. The blood is fluid or clotted, and there may be very characteristic moulds of the pelvis of the kidney and of the ureter. It would no doubt be possible for such to form in the hæmaturia from calculus, but I have never met with a case of blood casts of the pelvis and of the ureter, either alone or together, except in cancer. It is rare, indeed, that cancer elements can be recognized in the urine, and yet the diagnosis has been made in this way.

(*b*) Pain is an uncertain symptom. In several of the largest tumors

which have come under my observation there has been no discomfort from beginning to close. When present, it is of a dragging, dull character, situated in the flank and radiating down the thigh. The passage of the clots may cause great pain. In one case the growth was at first upward, and the symptoms for some months were those of pleurisy.

(*c*) Progressive emaciation. The loss of flesh is usually marked and advances rapidly. There may, however, be a very large tumor without emaciation.

Physical Signs.—In almost all instances tumor is present. When small and on the right side, it may be very movable; in some instances, occupying a position in the iliac fossa, it has been mistaken for ovarian tumor. The large growths fill the flank and gradually extend toward the middle line, occupying the right or left half of the abdomen. Inspection may show two or three hemispherical projections corresponding to distended sections of the organ. In children the abdomen may reach an enormous size and the veins are prominent and distended. On bimanual palpation the tumor is felt to occupy the lumbar region and can usually be lifted slightly from its bed; in some cases it is very movable, even when large; in others it is fixed, firm, and solid. The respiratory movements have but slight influence upon it. Rapidly growing renal tumors are soft, and on palpation may give a sense of fluctuation. A point of considerable importance is the fact that the colon crosses the tumor, and can usually be detected without difficulty.

Diagnosis.—In children very large abdominal tumors are either renal or retroperitoneal. The retroperitoneal sarcoma (Lobstein's cancer) is more central, but may attain as large a size. If the case is seen only toward the end, a differential diagnosis may be impossible; but, as a rule, the sarcoma is less movable. It is to be remembered that these tumors may invade the kidney. On the left side an enlarged spleen is readily distinguished, as the edge is very distinct and the notch or notches well marked; it descends during respiration, and the colon lies behind, not in front of it. On the right side growths of the liver are occasionally confounded with renal tumors; but such instances are rare, and there can usually be detected a zone of resonance between the upper margin of the renal tumor and the ribs. Late in the disease, however, this is not possible, for the renal tumor is in close union with the liver.

A malignant growth in a movable kidney may be very deceptive and may simulate cancer of the ovary or myoma of the uterus. The great mobility upward of the renal growth and the negative result of examination of the pelvic viscera are the reliable points.

When the growth is small and the patient in good condition removal of the organ may be undertaken, but the percentage of cases of recovery is very small, only 5.4 per cent. (G. Walker).

XIII. CYSTIC DISEASE OF THE KIDNEY

The following varieties of cysts are met with:

Small Cysts.—The small cysts, already described in connection with the chronic nephritis, which result from dilatation of obstructed tubules or of Bowman's capsules. There are cases very difficult to classify, in which the

kidneys are greatly enlarged, and very cystic in middle-aged or elderly persons, and yet not so large as in the congenital form.

Solitary Cysts.—Solitary cysts, ranging in size from a marble to an orange, or even larger, are occasionally found in kidneys which present no other changes. In exceptional cases they may form tumors of considerable size. Newman operated on one which contained 25 ounces of blood. They, too, in all probability, result from obstruction.

Polycystic Kidneys.—The polycystic kidneys, in which the greatly enlarged organs, weighing even as much as six pounds, are represented by a conglomeration of cysts, varying in size from a pea to a marble. Little or no renal tissue may be noticeable, although in microscopic sections it is seen that a considerable amount remains in the interspaces. The cysts contain a clear or turbid fluid, sometimes reddish brown or even blackish in color, and may be of a colloidal consistence. Albumin, blood crystals, cholesterin, with triple phosphates and fat drops, are found in the contents. Urea and uric acid are rarely present. The cysts are lined by a flattened epithelium. They occur in the fetus, and sometimes are of such a size as to obstruct labor. In the adult they are usually bilateral, and there is every reason to believe that they begin in early life and increase gradually. Indeed, a progressive growth has been noticed in some cases (Alfred King). They may be found in connection with cystic disease of the liver and other organs. It is difficult to account for the origin of this remarkable condition, which some regard as a defect of development rather than a pathological change, and point to the association in the fetal cases of other anomalies, as imperforate anus. Others believe the condition to be a new growth—a sort of mucoid endothelioma.

It is interesting to note that several members of a family may be affected. I have reported an instance in which mother and son were ~~the subjects of~~ the disease.

SYMPTOMS.—Of five cases which ~~I have seen~~ in adults the condition was recognized during life in four. The features are characteristic.

(*a*) Bilateral tumors in the renal regions, which may increase in size under observation. They may cause great enlargement of the upper zone of the abdomen. The colon and stomach are in front of the tumors, on the surface of which in very thin subjects the cysts may be palpable.

(*b*) Hæmaturia, which may recur at intervals for years.

(*c*) The signs of a chronic interstitial nephritis—(1) pallor or muddy complexion; in rare instances a bronzing of the skin; (2) sclerosis of the arteries; (3) hypertrophy of the heart with accentuated second sound; (4) urine abundant, of low specific gravity, with albumin, and hyaline and granular tube casts, and in one of ~~my~~ cases there were cholesterin crystals. Death occurs from uræmia or the cardio-vascular complications of chronic ~~Bright's~~ disease. A rare event is rupture of a cyst with the formation of a perinephric abscess and peritonitis. In two of ~~my~~ cases the skin became much pigmented.

While both kidneys are, as a rule, involved, one may be much smaller than the other.

Operation is rarely indicated, unless the condition is found to be unilateral, in which case Morris has removed the kidney in several instances, and the patients have remained well for years.

Other Varieties.—Occasionally the kidneys and liver present numerous small cysts scattered through the substance. The spleen and the thyroid also may be involved, and there may be congenital malformation of the heart. The cysts in the kidney are small, and neither so numerous nor so thickly set as in the conglomerate form, though in these cases the condition is probably the result of some congenital defect. There are cases, however, in which the kidneys are very large. It is more common in the lower animals than in man. I have seen several instances of it in the hog; in one case the liver weighed 40 pounds, and was converted into a mass of simple cysts. The kidneys were less involved. Charles Kennedy found references to 12 cases of combined cystic disease of the liver and kidneys.

The echinococcus cysts have been described under the section on parasites. Paranephric cysts (external to the capsule) are rare; they may reach a large size.

XIV. PERINEPHRIC ABSCESS

Suppuration in the connective tissue about the kidney may follow (1) blows and injuries; (2) the extension of inflammation from the pelvis of the kidney, the kidney itself, or the ureters; (3) perforation of the bowel, most commonly the appendix, in some instances the colon; (4) extension of suppuration from the spine, as in caries, or from the pleura, as in empyema; (5) as a sequel of the fevers, particularly in children.

Post mortem the kidney is surrounded by pus, particularly at the posterior part, though the pus may lie altogether in front, between the kidney and the peritoneum. Usually the abscess cavity is extensive. The pus is often offensive and may have a distinctly fæcal odor from contact with the large bowel. It may burrow in various directions and burst into the pleura and be discharged through the lungs. A more frequent direction is down the psoas muscle, when it appears in the groin, or it may pass along the iliacus fascia and appear at Poupart's ligament. It may perforate the bowel or rupture into the peritoneum; sometimes it penetrates the bladder or vagina.

Post mortem we occasionally find a condition of *chronic perinephritis* in which the fatty capsule of the kidney is extremely firm, with numerous bands of fibrous tissue, and is stripped off from the proper capsule with the greatest difficulty. Such a condition probably produces no symptoms.

Symptoms.—There may be intense pain, aggravated by pressure, in the lumbar region. In other instances the onset is insidious, without pain in the renal region; on examination signs of deep seated suppuration may be detected. On the affected side there is usually pain, which may be referred to the neighborhood of the hip joint or to the joint itself, or radiate down the thigh and be associated with the retraction of the testis. The patient lies with the thigh flexed, so as to relax the psoas muscle, and in walking throws, as far as possible, the weight on the opposite leg. He also keeps the spine immobile, assumes a stooping posture in walking, and has great difficulty in voluntarily adducting the thigh (Gibney).

There may be pus in the urine if the disease has extended from the pelvis or the kidney, but in other forms the urine is clear. When pus has formed there are usually chills with irregular fever and sweats. On examination,

deep seated induration is felt between the last rib and the crest of the ilium. Bimanual palpation may reveal a distinct tumor mass. Œdema or puffiness of the skin is frequently present.

Diagnosis.—The diagnosis is usually easy; when doubt exists the aspirator needle should be used. We can not always differentiate the primary forms from those due to perforation of the kidney or of the bowel. This, however, makes but little difference, for the treatment is identical. It is usually possible by the history and examination to exclude diseases of the vertebra. In children hip-joint disease may be suspected, but the pain is higher, and there is no fullness or tenderness over the hip-joint itself.

Treatment.—The treatment is clear—early, free, and permanent drainage.

SECTION VIII

DISEASES OF THE BLOOD

I. ANÆMIA

Anæmia, a reduction of the amount of blood as a whole or of its corpuscles, or of certain of its constituents, may be due to failure in the manufacture, to increase in the consumption, or to a sudden loss, as in hæmorrhage. Defective formation, hæmatogenesis, is responsible for a large group of what are known as the primary anæmias. Increased destruction, or hæmolysis, is the basis of the majority of all cases in which anæmia is secondary to some existing disease.

Anæmia may be local, confined to certain parts, or general, involving the entire body.

LOCAL ANÆMIA

Tissue irrigation with blood is primarily from the heart, but in all extensive systems of this sort provision is made at the local territories for variations in the supply, according to the needs of a part. The sluices are arranged by means of the stop-cock action of the arteries, which contract or expand under the influence of the vaso-motor ganglia, central and peripheral. If the sluices of one large district are too widely open, so much blood may enter that other important regions have not enough to keep them at work. Local anæmia of the brain, causing swooning, ensues when the mesenteric channels, capable of holding all the blood of the body, are wide open. Emotional stimuli, reflex from pain, etc., removal of pressure, as after tapping in ascites, may cause this. It is probable that many of the nervous and other symptoms in enteroptosis are due to the relative anæmia of the cerebral and spinal systems, owing to the persistent overfilling of the mesenteric reservoir. We know very little of local anæmia of the various organs, but possibly functional disturbance in the liver, kidneys, pancreas, heart, etc., may result from a permanently low pressure in the local blood "mains." Anæmia from spasm of the arterial walls is seen in Raynaud's disease, which usually affects the peripheral vessels, causing local syncope of the fingers, but it may occur in the visceral vessels, particularly of the brain, and cause temporary hemiplegia, aphasia, etc.

In local anæmia we are sometimes deceived by the appearance of the skin and mucous membranes. A marked pallor may exist with normal corpuscles and hæmoglobin; for example, the pallor after a drinking bout, or of nausea; in certain cases of heart disease, in lead-workers, and in the morphia habitué

the skin is often permanently pale. There are a few healthy people who are always pale, and yet have a practically normal blood count and color index.

GENERAL ANÆMIA

The general anæmias may be divided into the secondary or symptomatic, and the primary or essential.

Acute Secondary Anæmia

Etiology.—Hæmorrhage, certain acute infections, and intoxications are the important causes. A typical form is that which follows hæmorrhage, either traumatic or spontaneous. In rupture of a large vessel, or of an aneurism, in the peptic ulcer, or in injury to blood vessels the loss of three or four pounds of blood may prove fatal. Seven and a half pounds is the largest quantity I have known shed into one cavity (rupture of an aneurism into the pleura). A patient with hæmatemesis lost ten pounds of blood in one week, and yet recovered from the immediate effects. Even after the severest traumatic hæmorrhage the blood count is rarely so low as in certain forms of primary anæmia. Thus in the case of hæmatemesis just mentioned the red blood-corpuscles were 1,390,000 per c. mm.

Acute secondary anæmia may follow hæmolysis in certain infections, as malaria, acute endocarditis, sepsis, and a profound anæmia may be induced in the course of a week. Less often do we see an acute secondary anæmia follow toxic substances, such as mercury or nitro-benzol.

Symptoms.—Dyspnœa, rapid action of the heart, and faintness are the prominent symptoms of an acutely produced anæmia. There is marked pallor of the skin and mucous membranes, the pulse becomes small, the temperature is low, the patient feels giddy and faint and has noises in the ears. If the bleeding continues there may be nausea, vomiting, and, with the rapid loss of large quantities of blood, convulsions. Examination of the blood shows a great diminution of the red blood-corpuscles, often in severe hæmorrhage to two millions per c. mm. The hæmoglobin is proportionately lower, giving a color index of about 0.8. Irregularity in the red blood-corpuscles is seen; nucleated red corpuscles, usually normoblasts, appear early; the leucocytes are increased, usually the multi-nuclear neutrophiles. The process of regeneration goes on with great rapidity; the watery and saline constituents are readily restored by absorption; the albuminous elements are quickly renewed, but it may take weeks or months for the red blood-corpuscles to reach the normal standard. Thus in a case of purpura the red blood-corpuscles fell between the 20th and 30th April to below two millions, and the leucocytes rose to 12,000. It was not until July that the red blood-corpuscles reached four million, and the blood was not normal until September. The hæmoglobin is always restored more slowly than the corpuscles. This is very well illustrated in the accompanying chart (page 729).

In repeated hæmorrhages the picture depends upon the interval between the losses of blood. If long enough to allow of complete regeneration each time the total amount of blood lost may be very great. Ehrlich mentions the case of a patient with hæmoptysis who lost 20 kilograms of blood in 6½

book worm

months. If, however, the intervals are short, so that complete recovery from each loss of blood is not possible, a chronic anæmia is soon induced with a very watery plasma, a low color index, and lymphocytosis.

Chronic Secondary Anæmia

Etiology.—There are ~~very~~ many causes, of which ~~the following~~ are the most important:

(*a*) *Inanition.*—This may be brought about by defective food supply, or by conditions which interfere with the proper reception and preparation of

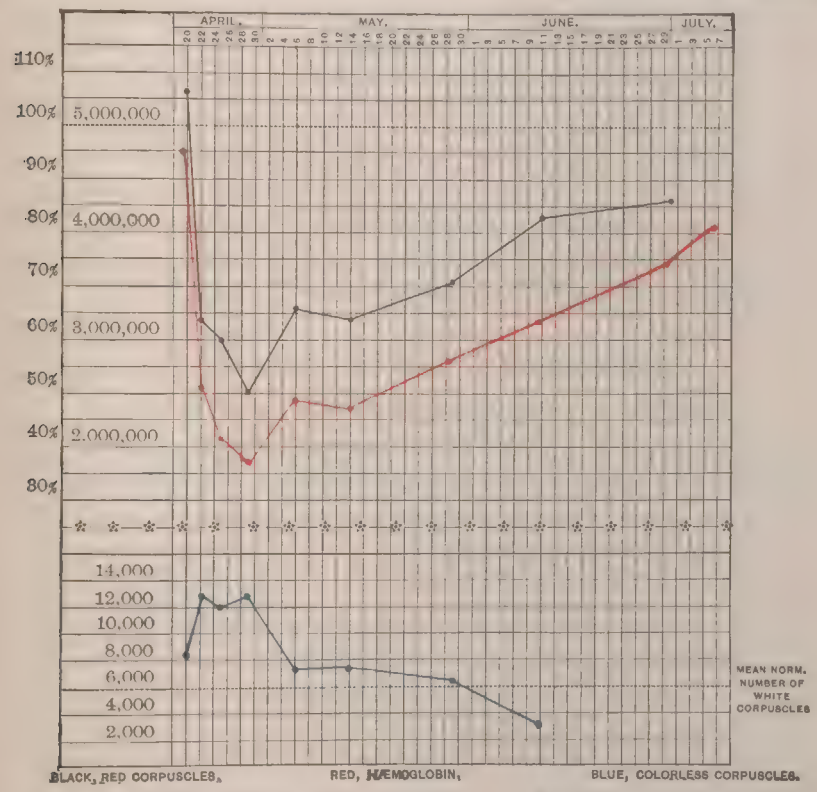

CHART XVI.—THE RAPIDITY WITH WHICH ANÆMIA IS PRODUCED IN PURPURA HÆMORRHAGICA, AND THE GRADUAL RECOVERY.

the food, as in cancer of the œsophagus and chronic dyspepsia. The reduction of the blood mass may be extreme, but the plasma suffers proportionately more than the corpuscles, which, even in the wasting of cancer of the œsophagus, may not be reduced more than one-half to three-fourths. The reduction in the plasma may be so great that the corpuscles show a relative increase.

(*b*) *Infections.*—In nearly all acute fevers anæmia is produced, which may persist after the infection has subsided. We see this particularly in typhoid fever, rheumatic fever, sepsis, syphilis, and malaria. Certain forms of animal parasites, as the anchylostoma and bothriocephalus, cause a profound anæmia.

(*c*) *Intoxications.*—Inorganic poisons, such as lead, mercury, arsenic; organic poisons, as the toxins of various fevers; and certain autogenous poisons occurring in chronic affections, such as nephritis and jaundice.

(*d*) *Hæmorrhage.*—This, if repeated, may cause severe anæmia. This is particularly shown in cases of persistent bleeding from hæmorrhoids.

(*e*) Long continued drain upon the system, as in chronic suppuration, prolonged lactation, and in rapidly growing tumors of all sorts.

Symptoms.—Loss of bodily and mental vigor with loss of weight and obvious anæmia are the important features. The patient tires easily, the appetite is poor, digestion often faulty, palpitation is complained of, and there may be feelings of faintness, and, as the anæmia progresses, swelling of the feet. There is not infrequently slight fever. Petechiæ on the skin are not uncommon, and retinal hæmorrhages may occur. The blood picture is distinctive. The red blood-corpuscles are reduced, but rarely below two millions per c. mm. In 59 cases of cancer of the stomach the average count was 3,712,186 per c. mm. In only 8 cases was the count below two millions, in none below one million per c. mm. The hæmoglobin is proportionately low in the cases just mentioned; the average was 44.9 per cent.; in only 9 cases was it below 30 per cent. The red blood-corpuscles are irregular in shape, nucleated forms are present, and the leucocytes are usually increased in number.

PRIMARY OR ESSENTIAL ANÆMIA

1. *Chlorosis*

Definition.—An anæmia of unknown cause, occurring in young girls, characterized by a marked relative diminution of the hæmoglobin.

Etiology.—It is a disease of girls, more often of blondes than of brunettes. It is doubtful if males are ever affected. The age of onset is between the fourteenth and seventeenth years; under the age of twelve cases are rare. Recurrences, which are common, may extend into the third decade. Of the essential cause of the disease we know nothing. There exists a lowered energy in the blood-making organs, associated in some obscure way with the evolution of the sexual apparatus in women. Hereditary influences, particularly chlorosis and tuberculosis, play a part in some cases. Sometimes, as Virchow pointed out, the condition exists with a defective development (hypoplasia) of the circulatory and generative organs.

The disease is most common among the ill-fed, overworked girls of large towns, who are confined all day in close, badly lighted rooms, or have to do much stair-climbing. Cases occur, however, under the most favorable conditions of life, but not often in country-bred girls, as Maudlin sings in the *Compleat Angler*. Lack of proper exercise and of fresh air and the use of improper food are important factors. Emotional and nervous disturbances may be prominent—so prominent that certain writers have regarded the disease as a neurosis. De Sauvages speaks of a *chlorose par amour*. Newly arrived Irish girls were very prone to the disease in Montreal. The "corset and chlorosis" expresses O. Rosenbach's opinion. Menstrual disturbances are not uncommon, but are probably a sequence, not a cause, of chlorosis. Sir Andrew Clark believed that constipation played an important rôle, and that

the condition is in reality a *copræmia* due to the absorption of poisons from the large bowel.

Symptoms.—(*a*) General.—The symptoms of chlorosis are those of anæmia. The subcutaneous fat is well retained or even increased in amount. The complexion is peculiar; neither the blanched aspect of hæmorrhage nor the muddy pallor of grave anæmia, but a curious yellow green tinge, which has given to the disease its name, and its popular designation, the green sickness. Occasionally the skin shows areas of pigmentation, particularly about the joints. In cases of moderate grade the color may be deceptive, as the

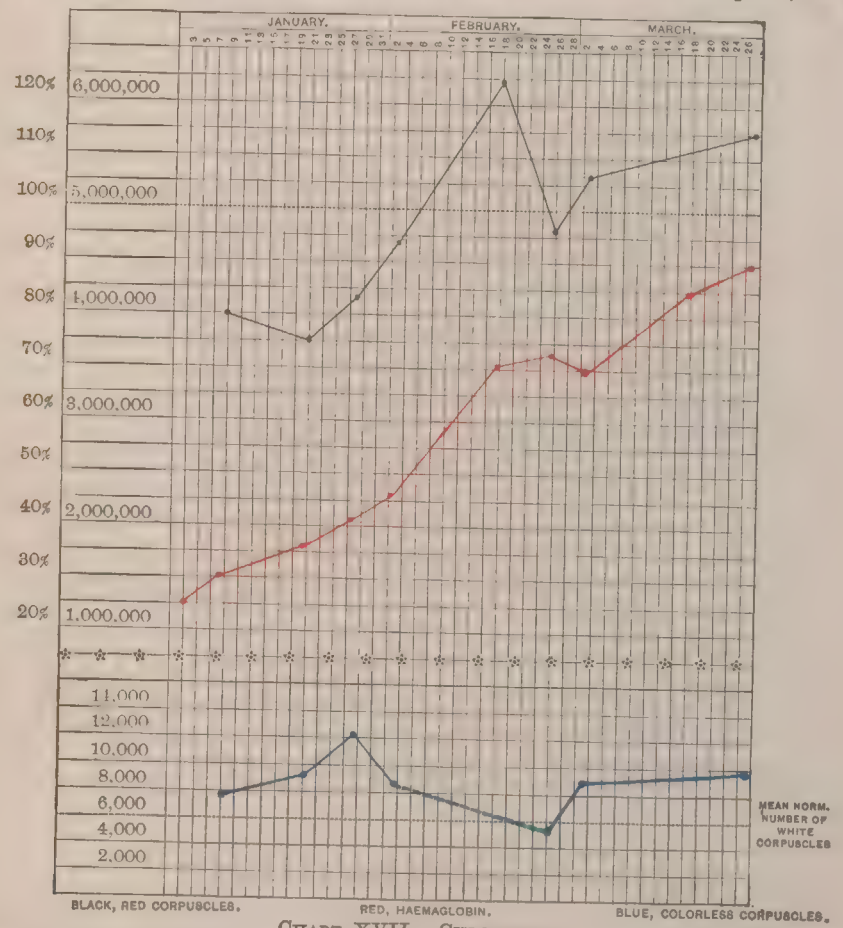

Chart XVII.—Chlorosis.

cheeks have a reddish tint, particularly on exertion (chlorosis rubra). The subjects complain of breathlessness and palpitation, and there may be a tendency to fainting—symptoms which often lead to the suspicion of heart or lung disease. Puffiness of the face and swelling of the ankles may suggest nephritis. The disposition often changes, and the girl becomes low-spirited and irritable. The eyes have a peculiar brilliancy and the sclerotics are of a bluish color.

(*b*) Special Features.—*Blood.*—The drop as expressed looks pale. Jo-

hann Duncan, in 1867, first called attention to the fact that the essential feature was not a great reduction in the number of the corpuscles, but a quantitative change in the hæmoglobin. The corpuscles themselves look pale. In 63 consecutive cases examined at my clinic by Thayer the average number per cubic millimetre of the red blood-corpuscles was 4,096,544, or over 80 per cent., whereas the percentage of hæmoglobin for the total number was 42.3 per cent. The accompanying chart illustrates well these striking differences. There may, however, be well-marked actual anæmia. The lowest blood-count in the series of cases referred to above was 1,932,000. There may be all the physical characteristics and symptoms of a profound anæmia with the number of the blood-corpuscles nearly at the normal standard. Thus in one instance the globular richness was over 85 per cent., with the hæmoglobin about 35. No other form of anæmia presents this feature, at least with the same constancy and in the same degree. The importance of the reduction in the hæmoglobin depends upon the fact that it is the iron-containing elements of the blood with which in respiration the oxygen enters into combination. This marked diminution in the iron has also been determined by chemical analysis of the blood. The microscopic characteristics of the blood are as follows: In severe cases the corpuscles may be extremely irregular in size and shape—poikilocytosis, which may occasionally be as marked as in some cases of pernicious anæmia. The large forms of red blood cells are not as common, and the average size is stated to be below normal. The color of the corpuscles is noticeably pale and the deficiency may be seen either in individual corpuscles or in the blood mixture prepared for counting. Nucleated red corpuscles (normoblasts) are not very uncommon, and may vary greatly in numbers in the same case at different periods. The leucocytes may show a slight increase; the average in the 63 cases above referred to was 8,467 per cubic millimetre.

(*c*) Gastro-intestinal Symptoms.—The appetite is capricious, and patients often have a longing for unusual articles, particularly acids. In some instances they eat all sorts of indigestible things, such as chalk or even earth. Superacidity of the gastric juice is common. Distress after eating and even cardialgic attacks may be present. Constipation is a common symptom, and, as already mentioned, has been regarded as an important element in causing the disease. A majority of chlorotic girls who wear corsets have gastroptosis, and on inflation the stomach will be found vertically placed; sometimes the organ is very much dilated. The motor power is usually well retained. Enteroptosis with palpable right kidney is not uncommon.

(*d*) Circulatory Symptoms.—Palpitation of the heart occurs on exertion, and may be the most distressing symptom of which the patient complains. Percussion may show slight increase in the transverse dulness. A systolic murmur is heard at the apex or at the base; more commonly at the latter, but in extreme cases at both. A diastolic murmur is rarely heard. The systolic murmur is usually loudest in the second left intercostal space, where there is sometimes a distinct pulsation. The exact mode of production is still in dispute. Balfour holds that it is produced at the mitral orifice by relative insufficiency of the valves in the dilated condition of the ventricle. On the right side of the neck over the jugular vein a continuous murmur may be heard, the *bruit de diable,* or humming-top murmur.

The pulse is usually full and soft. Visible impulse is present in the veins of the neck, as noted by Lancisi. Pulsation in the peripheral veins is sometimes seen. Thrombosis in the veins may occur, most commonly in the femoral, but occasionally in the cerebral sinuses. In 86 cases the veins of the legs were affected in 48, the cerebral sinuses in 29 (Lichtenstern). The chief danger in thrombosis of the extremities is pulmonary embolism, which occurred in 13 of 52 cases collected by Welch.

As in all forms of essential anæmia, fever is not uncommon. Chlorotic patients suffer frequently from headache and neuralgia, which may be paroxysmal. The hands and feet are often cold. Dermatographia is common. Hysterical manifestations are not infrequent. Menstrual disturbances are very common—amenorrhœa or dysmenorrhœa. With the improvement in the blood condition this function is usually restored.

Diagnosis.—The green sickness, as it is sometimes called, is in many instances recognized at a glance. The well-nourished condition of the girl, the peculiar complexion, which is most marked in brunettes, and the white or bluish sclerotics are very characteristic. A special danger exists in mistaking the apparent anæmia of the early stage of pulmonary tuberculosis for chlorosis. Mistakes of this sort may often be avoided by the very simple test furnished by allowing a drop of blood to fall on a white towel or a piece of blotting paper—a deficiency in hæmoglobin is readily appreciated. The palpitation of the heart and shortness of breath frequently suggest heart-disease, and the œdema of the feet and general pallor cause the cases to be mistaken for Bright's disease. In the great majority of cases the characters of the blood readily separate chlorosis from other forms of anæmia.

Tr. fr. h. 731

2. *Idiopathic or Pernicious Anæmia*

Definition.—A recurring and usually fatal anæmia of unknown origin, characterized by hæmolysis and imperfect action of the blood-making organs.

History.—Addison (1855) gave the first accurate account, and it is sometimes known as Addison's anæmia. Channing described cases of severe anæmia in the puerperal state. The writings of Gusserow and Biermer in the early seventies did much to awaken interest in the disease. The studies of Pepper (Secundus), H. C. Wood, and Palmer Howard made the disease very familiar to American and Canadian physicians. The special methods introduced by Ehrlich have greatly increased our knowledge of the state of the blood and the bone marrow in the disease.

Distribution.—It is a common and widespread disease. It was of frequent occurrence in Montreal; I saw many cases in Philadelphia and also at the Johns Hopkins Hospital, and it seems quite as common, or even more so, in England. As Cabot remarks, the incidence of the disease is a good deal a matter of keenness on the part of the practitioners of any district.

Etiology.—The figures here quoted are from Cabot's analysis of some 1,200 cases given in his article in my "System of Medicine." It is a disease of middle life; a great majority—922—occurred over the age of 36. The youngest patient I have seen was a boy of ten years. Two or three cases may occur in one family, as a father and two girls.

Of special etiological factors much stress has been laid upon pregnancy

and the puerperal state. Doubtless many of the patients reported by Channing and Gusserow do not belong in this group. Of the true disease it forms a very small fraction, according to Cabot only 18 among the 1,200 cases of his series.

Sex.—It is twice as common in males as in females, but it is slightly commoner in women under the age of 36.

Buccal and Gastro-intestinal Infection.—William Hunter, who has done so much good work on the subject, claims that a large number of cases grouped as pernicious anæmia are really of an infective nature, and not related to the true Addisonian anæmia, which he regards as a chronic infection due to ~~a specific glossitis with~~ oral, gastric, and intestinal sepsis. In a few cases there is a history of long standing diarrhœa.

Intestinal Parasites.—Anæmia of a very severe, and even of a pernicious, type may be induced by the bothriocephalus and by the hookworm.

Atrophy of the Stomach.—In a certain number of cases—61 in Cabot's series—there was atrophy of the gastro-intestinal mucosa, but it seems ~~not im~~probable, ~~as he suggests, that when these two diseases are associated~~ the atrophy is a result rather than a cause of the anæmia.

Hæmorrhage.—As a rule the anæmia which follows repeated hæmorrhages is of the secondary type with a very different blood picture, but in every long series of cases of Addison's anæmia there will be found a few with a history of bleeding piles, of recurring nose bleeding, or of repeated hæmorrhages from other sources.

Nervous shock and emotional strain have been present in a few instances.

We have not got much beyond the position of Addison, who characterized the disease which he was describing as "a general anæmia occurring without any discoverable cause whatever; cases in which there had been no previous loss of blood, no existing diarrhœa, no chlorosis, no purpura, no renal, splenic, myasmatic, glandular, strumous, or malignant disease."

Pathology and Morbid Anatomy.—The body is rarely emaciated. A lemon tint of the skin is present in a majority of the cases. The muscles often are intensely red in color, like horse flesh, while the fat is light yellow. Hæmorrhages are common on the skin and serous surfaces. The heart is usually large, flabby, and empty. ~~In one instance I obtained only 2 drachms of~~ blood from the right heart, and between 3 and 4 ~~from the left.~~ The muscle substance of the heart is intensely fatty, and of a pale, light yellow color. In no affection do we see more extreme fatty degeneration. The lungs show no special changes. The stomach in many instances is normal, but in some cases of fatal anæmia the mucosa has been extensively atrophied. In the case described by Henry and ~~myself~~, the mucous membrane had a smooth, cuticular appearance, and there was complete atrophy of the secreting tubules. The liver may be enlarged and fatty. ~~In most of my autopsies it was normal in size, but usually fatty~~. The iron is in excess, a striking contrast to the condition in cases of secondary anæmia. It is deposited in the outer and middle zones of the lobules.

The spleen shows no important changes. In one of Palmer Howard's cases the organ weighed only 1 ounce and 5 drachms. The iron pigment is usually in excess. The lymph glands may be of a deep red color (hæmo-lymph gland). The amount of iron pigment is increased in the kidneys, chiefly in

the convoluted tubules. The bone-marrow is usually red, lymphoid in character, showing great numbers of nucleated red corpuscles, especially the larger forms called by Ehrlich gigantoblasts. There are cases in which the bone-marrow shows no signs of activity—*aplastic anæmia.*

Spinal cord lesions were present in 84 per cent. of the post mortems collected by Cabot. They affect chiefly the posterior columns of the cervical region.

The exact nature of the disease is unknown. Two views prevail, one that it is a hæmolysis produced by poisons intestinal or metabolic. Bunting has shown that a picture very similar to that of pernicious anæmia may be produced experimentally in animals by the injection of small doses of ricin. The investigations of Schaumann and others have shown the bothriocephalus anæmia to be a hæmolysis caused by a lipoid substance that may be extracted from the segments of the worm. From the intestinal mucosa of persons dead of pernicious anæmia lipoid substances have been extracted with hæmolytic action of remarkable potency, causing anæmia of a severe and fatal type in animals. These are interesting and suggestive facts, the only ones I know in favor of a special hæmolytic body. The majority of patients with pernicious anæmia have good appetites and good digestion, and usually get well from the first and second attacks without any special change in the condition of the bowels.

The view has been put forward by Moffitt and others that pernicious anæmia is an infection, possibly protozoal in character, in favor of which are the facts that anæmia is a very striking feature in many protozoal diseases, the occurrence of fever, the remarkable remissions, the nervous lesions, and the value of arsenic in treatment.

Symptoms.—The first thing that often attracts attention in a case of pernicious anæmia is the combination of pallor with good nutrition. As a rule there is very slight loss in weight and the fat layer is well preserved, so that the condition offers a striking contrast to most of the secondary anæmias, with which wasting is associated. The description given by Addison presents the chief features of the disease in a masterly way: "It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitations are produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking

contrast to the failure and exhaustion observable in every other respect."

A surprising fact is that there are patients with extreme anæmia who are remarkably vigorous, I recently saw a powerfully built man with 2,300,000 red blood-corpuscles per c. mm., who insisted that he was able to do everything as usual except that he was a little short of breath.

The appearance of the patient is usually very characteristic. The combination of a lemon-yellow tint of the skin with retention of the fat gives a very suggestive picture. Sometimes the tint is icteroid. In rare cases there is a white, anæmic pallor, and in a third group a brownish tinge of the skin (which is sometimes associated with leucoderma) deep enough to suggest Addison's disease. Muscular weakness, palpitation, headache, dyspnœa, vertigo, and œdema of the feet are common in this as in other types of anæmia.

Gastro-intestinal symptoms are not uncommon. Paroxysms of pain in the stomach with or without diarrhœa may occur in crises. In fully one-half of the cases diarrhœa occurs at some time during the course. The hydrochloric acid is usually greatly diminished or absent, and there may be complete achylia. A sore mouth and tongue, a feature to which attention was called especially by William Hunter, has not been common in my experience. There may be marked glossitis and ulcers. Pyorrhœa alveolaris may be said to be present in all cases, and the teeth are often very bad. Not infrequently the patients come for palpitation and disturbance of the heart. Slight dilatation is common; murmurs are rarely missed, generally hæmic and basic. Apex diastolic murmurs may occur without valve lesions. Extraordinary throbbing of the arteries may occur, so that aneurism may be suspected; the pulse may be collapsing. Œdema is common, usually in the feet, sometimes in the hands.

The urine is usually of low specific gravity, pale, and with diminished pigments. Sometimes, as pointed out by Hunter and Mott, it is of a deep sherry color, due to great excess of urobilin.

Nervous Symptoms.—Numbness and tingling are common, and sometimes there are marked neuritic pains. Anatomically it has been shown that lesions of the spinal cord are not at all uncommon. There are three groups of cases:

(*a*) The patient may have had no special symptoms pointing to involvement of the nervous system, but post mortem well marked lesions of the cord are found.

(*b*) With the anæmia there are signs of spinal cord lesions, either of a lateral sclerosis with spastic features and increased reflexes, or the picture may be rather of the tabetic type—lightning pains, girdle sensation, areas of anæsthesia, loss of the reflexes.

(*c*) There is a remarkable group carefully described by Risien Russell, Batten, and Collier, in which the nervous symptoms, usually those of a postero-lateral sclerosis, precede the anæmia.

As the disease progresses there may be great depression, sometimes delusions, but mental symptoms, as a rule, are not marked.

Hæmorrhages are not very uncommon, chiefly in the form of small petechiæ in the skin. Retinal hæmorrhages are frequent. Optic neuritis is very rare.

Blood.—The total quantity ~~in the body~~ is much diminished. The drop may look of good color, but it is abnormally fluid. The red blood-corpuscles are greatly diminished; the average count in 81 cases, when they came under observation, was 1,575,000 per c. mm. As Cabot says, there is no other disease which so often reduces the number of red blood-corpuscles below two millions per c. mm. In 12 per cent. of ~~my~~ cases the count was under one million. The lowest count on record is in a patient of Quincke's, 143,000 per c. mm.

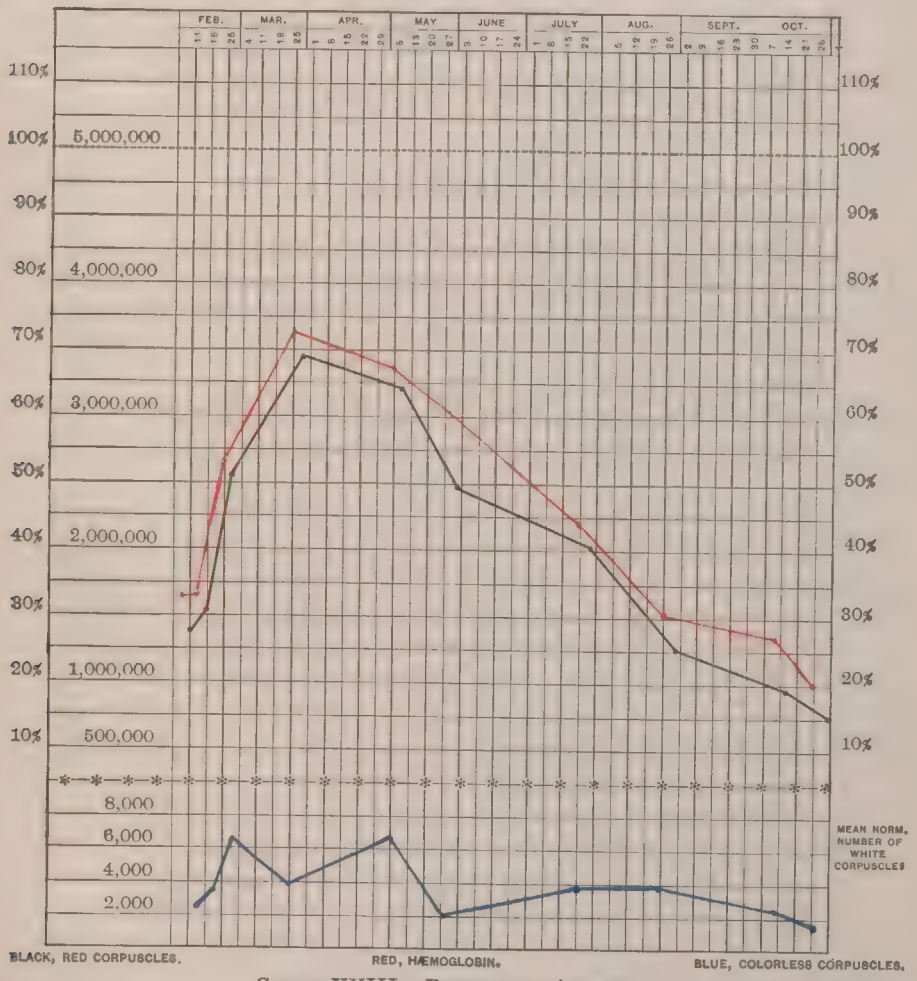

CHART XVIII.—PERNICIOUS ANÆMIA.

The hæmoglobin, though quantitatively reduced, is relatively high. This is one of the most constant and distinctive features of the disease, connected probably with an average increase in the size of the red blood-corpuscles. Stained films of the blood show great variation in the shape of the red blood-corpuscles—poikilocytosis. There are large giant forms, megalocytes, ovoid, measuring 8, 11, or even 15 μ. As Laache showed, this is one of the most pathognomonic features of the disease. On the other hand, there are a great

many very small red corpuscles—microcytes, from 2 to 6 μ in diameter, and of a deep red color. The irregularity in the shape of the red corpuscles is remarkable. Some are elongated, rod-like, others pyriform; one end of the corpuscle may be of normal shape, while the other is extended like the neck of a bottle. Stippling of the red blood-corpuscles is common with dark blue or blackish discoloration—the so-called polychromatophilia.

Nucleated red blood-corpuscles are constantly present, varying very much in numbers from day to day. There are two types—normoblasts of the average size, and the megaloblasts, which are much larger. There are frequently intermediate forms between these two groups. These nucleated red cells vary extraordinarily in different cases, and there may be what have been called blood crises, in which a large number of the nucleated reds appear. In one such crisis there were 14,388 normoblasts, 460 intermediates, and 138 megaloblasts per c. mm. These crises are sometimes followed by gains in the blood count, but they may be terminal events, and not specially indicative of active blood regeneration.

The leucocytes are generally normal or diminished in number. Polynuclear cells are rarely reduced. Occasionally there is a marked increase in the small mononuclear forms. Myelocytes are frequently present, even up to 8 and 10 per cent. Blood-platelets are usually low; counts of 100,000 and less are not uncommon (Pratt).

Chart XVIII ~~gives a very good idea of~~ the blood condition in a case during nine months.

Aplastic Anæmia.—A certain number of cases of primary anæmia run a rapid and progressive course, without remissions; and death occurs within a few months from the beginning of the attack. Post mortem, instead of an active hyperplasia of the bone marrow, there is atrophy or aplasia. To these cases the term "aplastic anæmia" has been given. It is a sub-type of pernicious anæmia with identical clinical features, except that it runs a more rapid course, is met with in younger persons, the color index may be low, hæmorrhages are more common, there may be leucopenia, and erythroblasts are usually absent. The hæmorrhages may be very severe, and some of the cases are of a pronounced purpuric type.

The diagnosis is only certain after an examination of the bones, when it is found that the marrow of the long bones is fatty, and even the red marrow may have disappeared from the short bones.

Forms of splenic anæmia, suggesting the primary pernicious form, and leukanæmia will be discussed elsewhere.

Prognosis and Course.—The disease sometimes runs a very acute course. In a patient seen with Finley in Montreal the fatal termination occurred within ten days of the onset of the symptoms. In other cases the course is from six to twelve weeks, but, as a rule, it is a chronic malady with remarkable remissions. It is rare to meet with a case in which recovery does not take place from the first attack. The number of remissions varies from two or three to five or six. In 521 cases analyzed by Cabot for this special point, 296 had one remission, 118 two, 65 three, 21 four, and 24 five. The duration of the remission may be from three months to four years. In 81 cases ~~treated in my wards death~~ occurred in 27 while under observation. The average duration in these cases was about a year.

no evidence of siemens blood distinction
Plato on demens marketing

nephritis

The ultimate prognosis in a great majority of cases is bad; only one case in our series appears to have recovered completely, another was alive and in good health six years after the last attack, and a third four years after. In Cabot's series there were ten cases which had lasted seven years or more, but there were only 6 out of the 1,200 cases analyzed which he regarded as having completely recovered.

Diagnosis.—Few diseases are more readily recognized at sight. There is something very characteristic about the general appearance of a patient with Addisonian anæmia, and nowadays practitioners are much more alert, and the disease is better known. The lemon colored tint of the skin may suggest jaundice; the anæmia, puffy face, swollen ankles, and albumin in the urine, ~~Bright's disease~~; the pigmentation, Addison's disease; the shortness of breath and palpitation, heart disease; the pallor and gastric symptoms, cancer of the stomach. The retention of fat, the insidious onset, the absence of signs of local disease, and the blood features ~~already discussed~~ are the important diagnostic points. From cancer of the stomach pernicious anæmia is distinguished by the absence of wasting, the high color index of the blood, the lower corpuscular count, and by the marked improvement in the first attacks under proper treatment.

~~TREATMENT OF ANÆMIA~~ To h. 730,

Secondary Anæmia.—The traumatic cases do best, and with plenty of good food and fresh air the blood is readily restored. The extraordinary rapidity with which the normal percentage of red blood-corpuscles is reached without any medication whatever is an important lesson. The cause of the hæmorrhage should be sought and the necessary indications met. The large group depending on the drain on the albuminous materials of the blood, as in ~~Bright's disease~~, suppuration, and fever, is difficult to treat successfully, and so long as the cause keeps up it is impossible to restore the normal blood condition. The anæmia of inanition requires plenty of nourishing food. When dependent on organic changes in the gastro-intestinal mucosa not much can be expected from either food or medicine. In the toxic cases due to mercury and lead the poison must be eliminated and a nutritious diet given with full doses of iron. In a great majority of these cases there is deficient blood formation, and the indications are briefly three: plenty of food, an open-air life, and iron. As a rule, it makes but little difference what form of the drug is administered. In severe forms the patient should be at rest in bed and in the open air, if possible.

Chlorosis.—The treatment of chlorosis affords one of the most brilliant instances—of which we have but three or four—of the specific action of a remedy. Apart from the action of quinine in malarial fever, and of mercury and iodide of potassium in syphilis, there is no other drug the beneficial effects of which we can trace with the accuracy of a scientific experiment. It is a minor matter *how* the iron cures chlorosis. In a week we give to a case as much iron as is contained in the entire blood, as even in the worst case of chlorosis there is rarely a deficit of more than 2 grams of this metal. Iron is present in the fæces of chlorotic patients before they are placed upon any treatment, so that the disease does not result from any deficiency of available

iron in the food. Bunge believes that it is the sulphur which interreres with the digestion and assimilation of this natural iron. The sulphides are produced in the process of fermentation and decomposition in the fæces, and interfere with the assimilation of the normal iron contained in the food. By the administration of an inorganic preparation of iron, with which these sulphides unite, the natural organic combinations in the food are spared.

In studying charts of chlorosis, it is seen that there is an increase in the red blood-corpuscles under the influence of the iron, and in some instances the globular richness rises above normal. The increase in the hæmoglobin is slower and the maximum percentage may not be reached for a long time. I have for years in the treatment of chlorosis used with the greatest success Blaud's pills, made and given according to the formula in Niemeyer's text-book, in which each pill contains 2 grains of the sulphate of iron. During the first week one pill is given three times a day; in the second week, two pills; in the third week, three pills, three times a day. This dose should be continued for four or five weeks at least before reduction. An important feature in the treatment is to persist in the use of the iron for at least three months, and, if necessary, subsequently to resume it in smaller doses, as recurrences are so common. The diet should consist of good, easily digested food. Special care should be directed to the bowels, and if constipation is present a saline purge should be given each morning. Such stress did Sir Andrew Clark lay on the importance of constipation in chlorosis that he stated that if limited to the choice of one drug in the treatment of the disease he would choose a purgative. In many instances the dyspeptic symptoms may be relieved by alkalies. Dilute hydrochloric acid, manganese, phosphorus, and oxygen have been recommended. Rest in bed is important in severe cases.

Pernicious Anæmia.—There are five essentials: first, a diagnosis; secondly, rest in bed for weeks or even months, if possible (thirdly) in the open air; fourthly, all the good food the patient can take; the outlook depends largely on the stomach; fifthly, arsenic; Fowler's solution in increasing doses, beginning with ♏ iii or v (0.2 to 0.3 c. c.) three times a day, and increasing ♏ i each week until the patient takes ♏ xv (1 c. c.) three times a day. Other forms of arsenic may be tried, as the sodium cacodylate or the atoxyl hypodermically. Atoxyl can be given in doses of gr. ss (0.032 gm.) every five days, and the amount gradually increased. Accessories are oil inunctions; bone-marrow, which has the merit of a recommendation by Galen; in some cases iron seems to do good. Care should be taken of the mouth and teeth. Gastric lavage and irrigations of the colon are useful in some cases.

Injections of blood serum and defibrinated blood have been given. The serum is given in small amounts, 10 to 20 c. c., usually into a vein; rabbit serum is perhaps the best. Defibrinated human blood should be given intravenously in large amounts, up to 500 c. c. This is better than to attempt direct transfusion. It is important to test the blood so that there is certainty of using a homologous serum (see Moss, *Johns Hopkins Hosp. Bull.*, 1911, xxii, p. 238). This treatment should only be carried out by those who are familiar with the problems involved and is not advisable for general use.

After recovery the patient should be told to watch the earliest indications of return of the trouble and at once resume the arsenic.

Splenectomy has been done in a number of cases but it is still too early to judge of its value. Some patients have been helped for a time but that permanent benefit results is not yet proved.

II. LEUKÆMIA

Definition.—A disease characterized by a permanent increase in the leucocytes of the blood, associated with hyperplasia of the leucoblastic tissues.

History.—In October, 1845, Hughes Bennett recorded a case of "suppuration of the blood with enlargement of the spleen and liver," and he afterward gave the disease the name of "leukocythæmia." A month later Virchow described a similar condition of "white blood" to which he gave the name of "leukæmia." In 1870 Neumann determined the importance of the changes in the bone marrow in connection with the disease.

Varieties.—The whole hæmatopoietic system—marrow, spleen, and lymph glands—is involved ~~in the disease~~. Formerly we spoke of three different groups—the splenic, lymphatic, and medullary, but we now recognize that the leucoblastic hyperplasia may begin in any part of the blood-glandular system, marrow, lymph glands, and probably in the spleen. The differences in the types ~~of the disease~~ depend upon the dominance of the lymphoid or the myeloid process, so that we now divide the cases roughly into two great groups: (1) the myelocytic or myeloid, corresponding to the spleno-medullary type, and (2) the lymphoid, which represents the lymphatic variety. Some cases not fitting accurately into either are spoken of as "atypical" or "transitional" forms.

The nature of the disease is unknown. The acutely fatal cases resemble an infection, but no organisms have been determined. Banti, Warthin, and others regard the disease as related to myeloma and sarcoma.

I. Myeloid Leukæmia.—*Etiology.*—The disease is not very rare. There were 24 cases in ~~my~~ wards at the Johns Hopkins Hospital in fifteen years. It certainly is not more frequent in malarial regions.

It is rather more common in males than in females, and between the 30th and 50th years. The youngest of ~~my~~ patients was a child of eight months.

In some instances it has followed a blow. Some of the patients have had a tendency to hæmorrhage, but, as a rule, the disease appears in fairly healthy persons without any recognizable cause. It may occur during pregnancy, and a leukæmic patient of Cameron's of Montreal passed through three pregnancies, bearing on each occasion a non-leukæmic child. One of this patient's children had leukæmia before the mother showed signs of the disease, and another died of it. This patient's grandmother, mother, and brother suffered from symptoms strongly suggestive of leukæmia.

Morbid Anatomy.—Dropsy is sometimes present. There is in many cases a condition of polyæmia; the heart and veins are distended with large blood-clots. In ~~Case XI of my series~~, the weight of blood in the heart chambers alone was 620 grams. There may be remarkable distention of the portal, cerebral, pulmonary, and subcutaneous veins. The blood is usually clotted, and the enormous increase in the leucocytes gives a pus like appearance to the coagula, so that it has happened more than once, as in Virchow's memorable case, that on opening the right auricle the observer at first thought he had cut into an abscess. The coagula have a peculiar greenish color, somewhat like the fat of a turtle. Sometimes this is so intense as to suggest the color of chloroma, described later. ~~The alkalinity of the blood~~ is diminished.

The fibrin is increased. Charcot's octahedral crystals may separate from the blood after death.

In the myelitic form the spleen is greatly enlarged, the capsule may be thickened, and the vessels at the hilus enlarged. The weight may range from 2 to 18 pounds. The organ is in a condition of chronic hyperplasia. It cuts with resistance, has a uniformly reddish brown color, and the Malpighian bodies are invisible. Grayish white, circumscribed, lymphoid tumors may occur throughout the organ, contrasting strongly with the reddish brown matrix. Instead of a fatty tissue, the medulla of the long bones may resemble the consistent matter which forms the core of an abscess, or it may be dark brown in color. There may be hæmorrhagic infarctions. There may be much expansion of the shell of bone, and localized swellings which are tender and may even yield to firm pressure.

In some instances there are leukæmic enlargements in the solitary and agminated glands of Peyer. In a case of Willcocks' there were growths on the surface of the stomach and gastro-splenic omentum. The thymus is rarely involved, though it has been enlarged in some of the acute cases. The liver may be greatly enlarged, due to a diffuse leukæmic infiltration. There may be definite leukæmic growths. There are rarely changes of importance in the lungs. ~~The kidneys are often enlarged and pale, the capillaries may be distended with leucocytes, and leukæmic tumors may occur~~. The skin may present leukæmic tumors.

Leukæmic tumors in the organs are not common. In 159 cases collected by Gowers there were ~~only~~ 13 instances of leukæmic nodules in the liver and 10 in the kidneys.

Symptoms.—The onset is insidious, and, as a rule, the patient seeks advice for progressive enlargement of the abdomen and shortness of breath, or the pallor, palpitation, and other symptoms of anæmia. Bleeding at the nose is common. Gastro-intestinal symptoms may precede the onset. Occasionally the first symptoms are of a very serious nature. In one of the cases of my series the boy played lacrosse two days before the onset of the final hæmatemesis; and in another case a girl, who had, it was supposed, only a slight chlorosis, died of fatal hæmorrhage from the stomach before any suspicion had been aroused as to the true condition.

Anæmia is not a necessary accompaniment of all stages of the disease; the subjects may look very healthy and well.

The gradual increase in the volume of the spleen is the most prominent feature in a majority of the cases. Pain and tenderness are common, though the progressive enlargement may be painless. A creaking fremitus may be felt on palpation. The enlarged organ extends downward to the right, and may be felt just at the costal edge, or when large it may extend as far over as the navel. In many cases it occupies fully one half of the abdomen, reaching to the pubes below and extending beyond the middle line. As a rule, the edge, in some the notch or notches, can be felt distinctly. Its size varies greatly from time to time. It may be perceptibly larger after meals. A hæmorrhage or free diarrhœa may reduce the size. The pressure of the enlarged organ may cause distress after eating; in one case it caused fatal obstruction of the bowels. On auscultation a murmur may sometimes be heard over the spleen, and Gerhardt has described a pulsation in it.

The pulse is usually rapid, soft, compressible, but often full in volume. There are rarely any cardiac symptoms. The apex beat may be lifted an interspace by the enlarged spleen. Toward the close œdema may occur in the feet or general anasarca. Hæmorrhage is common. There may be most extensive purpura, or hæmorrhagic exudate into pleura or peritoneum. Epistaxis is the most frequent form. Hæmoptysis and hæmaturia are rare. Bleeding from the gums may be present. Hæmatemesis proved fatal in two of my cases, and in a third a large cerebral hæmorrhage rapidly killed. The leukæmic retinitis is a part of the hæmorrhagic manifestations. J. Hughes Bennet's first leukæmic patient died suddenly, without obvious cause.

Local gangrene may develop, with signs of intense infection and high fever. There are very few pulmonary symptoms. The shortness of breath is due, as a rule, to the anæmia. Toward the end there may be œdema of the lungs, or pneumonia may carry off the patient. The gastro-intestinal symptoms are rarely absent. Nausea and vomiting are early features in some cases, and diarrhœa may be very troublesome, even fatal. Intestinal hæmorrhage is not common. There may be a dysenteric process in the colon. Jaundice rarely occurs, though in one case of my series there were recurrent attacks. Ascites may be a prominent symptom, probably due to the presence of the splenic tumor. A leukæmic peritonitis also may be present, due to new growths in the membranes.

The nervous system is not often involved. Facial paralysis has been noted. Headache, dizziness, and fainting spells are due to anæmia. The patients are usually tranquil. Coma may follow cerebral hæmorrhage.

The special senses are often affected. There is a peculiar retinitis, due chiefly to the extravasation of blood, but there may be aggregations of leucocytes, forming small leukæmic growths. Optic neuritis is rare. Deafness has frequently been observed; it may appear early and possibly is due to hæmorrhage. Features suggestive of Ménière's disease may come on quite suddenly, due to leukæmic infiltration or hæmorrhage into the semi-circular canal.

The urine presents no constant changes. The uric acid excreted is always in excess.

Priapism is a curious symptom which has been present in a large number of cases. It may, as in one of our patients, be the first symptom. In one of my patients it persisted for seven weeks. The cause is not known.

Fever was present in two-thirds of my series. Periods of pyrexia may alternate with prolonged intervals of freedom. The temperature may range from 102° to 103° F.

Blood.—In all forms of the disease the diagnosis must be made by the examination of the blood, as it alone offers distinctive features.

The striking change is an increase in the colorless corpuscles. The average in my series was 298,700 per c. mm., and the average ratio to the red cells was 1 to 10. Counts above 500,000 per c. mm. are common, and they may rise above 1,000,000 per c. mm. The proportion of white to red cells may be 1 to 5, or may even reach 1 to 1. There are instances on record in which the number of leucocytes has exceeded that of the red corpuscles.

The increase is in all the forms. The polynuclear neutrophiles make up from 30 to 50 per cent.; both the small and the large lymphocytes are increased; the eosinophiles and the mast cells show both a percentage and ab-

solute increase. The abnormal cells, the myelocytes, range from 30 to 50 per cent. Normoblasts and megaloblasts are common. There is no anæmia at first. The red cell count may be normal, but sooner or later anæmia comes on, and the count may fall to 2,000,000 per c. mm. The color index is usu-

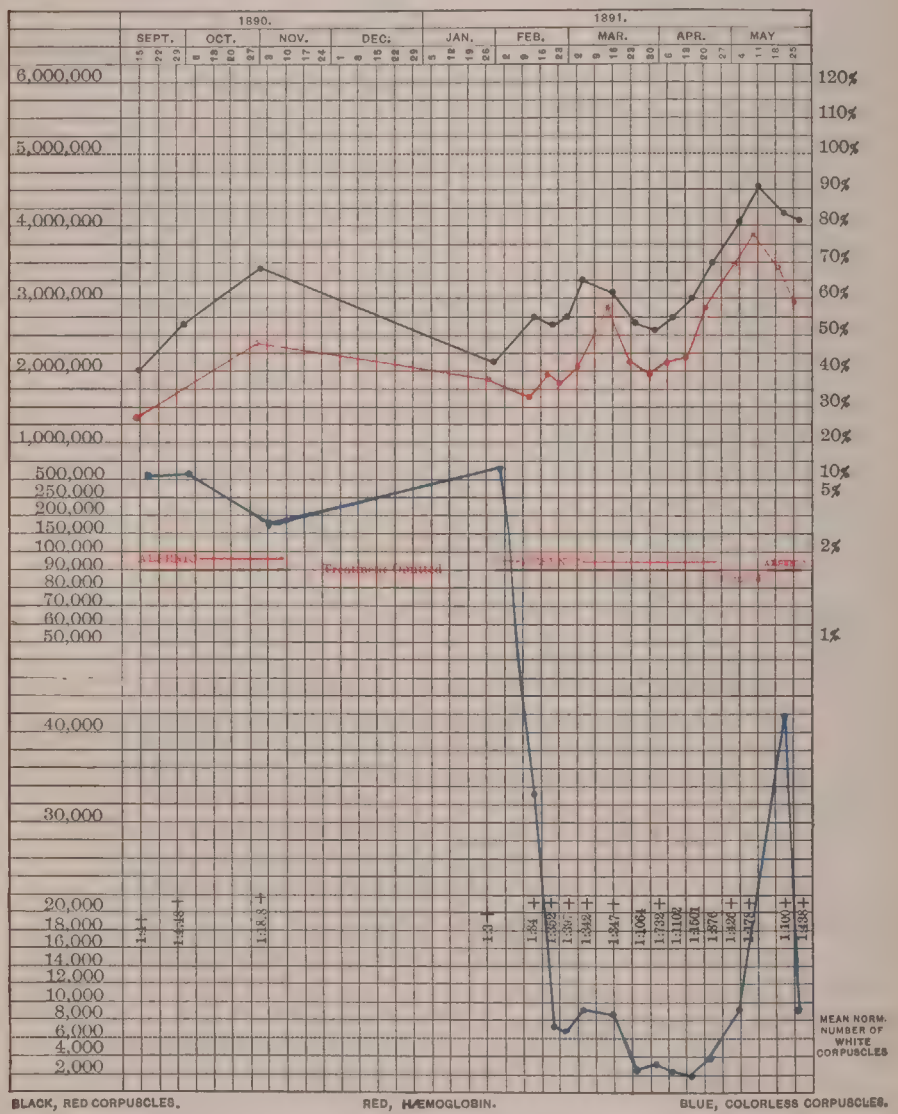

CHART XIX.—LEUKÆMIA.

ally low. The blood platelets are increased. Charcot-Leyden crystals may separate from the clots and the hæmoglobin shows a remarkable tendency to crystalize.

II. LYMPHOID LEUKÆMIA.—*Symptoms.*—This less common form occurs more frequently in males. There are two varieties, the acute and the chronic.

No. of leucocytes.

Acute lymphatic leukæmia, one of the most terrible of all the blood diseases, may run a malignant course unparalleled by any of them. Among the early symptoms are angina, often of an ulcerative character, involving the tonsils and the pharynx. Hæmorrhages occur early, usually into the skin, or there is profuse nose-bleeding. The swelling of the glands, most commonly of the neck, is then noticed; the patient rapidly becomes anæmic, and death has occurred as early as the seventh day. There may be marked fever, to 103° or 105° F., and the case may be mistaken for malignant typhoid or typhus fever. The real nature of the disease may not be evident until the lymph glands begin to swell; in some cases there is no evident glandular enlargement.

The chronic form is a very different disease, occurring later in life, and beginning with a general enlargement of the lymph glands, first the cervical, then the axillary. The spleen may be slightly enlarged, and anæmia eventually comes on, but the disease may last for years without any special anæmia. Hæmorrhages are rare. Fever is not common. A pruritus of great intensity may be present, sometimes with ecthymatous patches. The skin may become deeply pigmented. Localized leukæmic tumors of the skin have been described.

Blood.—The drop may be sticky and viscous, spreading with difficulty. The most remarkable feature is the increase of lymphocytes, which are very often above 90 per cent., and may reach even 99 per cent. There are two chief forms, the large and the small mononuclear. The majority of the chronic cases have the small lymphocytes, and the total percentage is rarely so high. Large lymphocytes are now thought to come from the bone marrow, but there is no sharp distinction between the forms, and possibly *all* have this origin.

Atypical Leukæmias.—(1) *Mixed leukæmias,* in part myeloid and in part lymphoid; but in nearly all cases of the ordinary spleno-medullary leukæmia a certain percentage of lymphocytes are present, which toward the end may be materially increased.

(2) *Cases with atypical blood changes,* such as a very high percentage of eosinophiles, or a condition with a very high proportion of plasma cells.

(3) *Chloroma* is an atypical lymphoid leukæmia in which the lymphatic tumors have a greenish color. The tumor growths occur chiefly in the skull, the orbit, the long bones, and throughout the viscera. The blood picture is like that of leukæmia and the condition is generally fatal within six or eight months.

(4) In a few rare instances a condition of leukæmia has been found without changes in the blood-making organs.

(5) *Leukanæmia.*—This term was invented by Leube to describe a condition showing features both of leukæmia and severe anæmia. The cases are now regarded as a myeloid leukæmia with severe anæmia. Glandular enlargement is usually present; the onset may be like the acute types of leukæmia, and the blood picture may be either of the lymphoid or of the myeloid type.

Diagnosis.—The recognition of the acute forms may be difficult, particularly those which begin with marked angina and cutaneous hæmorrhages. It may not be until a blood examination is made or the glands enlarge that

suspicion is aroused. The chronic forms are easily recognized. The enlarged spleen at once suggests a blood count, upon which alone the diagnosis rests. Twice I have had cases of leukæmia sent from the ophthalmic surgeon, one case with the diagnosis. In the lymphatic form, too, the diagnosis rests with the blood examination. One has to recognize that there are certain cases of sepsis with marked lymphocytosis, in which the white blood-corpuscles may reach 30,000 or 40,000 per c. mm. When the regional lymph glands are involved this may raise a doubt. Cabot gives an instance of a child in whom after pneumonia and whooping-cough there was a leucocytosis of 94,000 per c. mm. It is important to remember that in the ordinary myelitic forms under treatment with arsenic or with X-rays the increase of leucocytes may disappear, but the differential count may still be characteristic.

Prognosis.—Recovery in leukæmia is practically unknown. The acute cases die within three months; the chronic forms last from six months to four or five years. The chronic lymphatic form seems to be the most protracted. One case in my hospital series lasted three years. A private patient with accurate blood counts, in whom the diagnosis was made by W. H. Draper, was seen by me ten years subsequently; the cervical, axillary, and inguinal glands were greatly enlarged; the leucocytes were 242,000 per c. mm., above 90 per cent. being lymphocytes.

Association with Other Diseases.—Tuberculosis is not uncommon. Dock has collected 27 cases, in none of which did the tuberculosis show any special influence. Intercurrent infections are not rare, such as influenza, erysipelas, or sepsis—often with a remarkable effect upon the disease. In a case reported by Dock, after an attack of influenza the leucocytes fell from 367,000 to 7,500 per c. mm. Various other conditions influence the disease, and the leucocytes have disappeared under the use of arsenic, quinine, tuberculin, and the X-rays.

Treatment.—Fresh air, good diet, and abstention from mental worry and care are the important general indications. The *indicatio morbi* can not be met. There are certain remedies which have an influence upon the disease. Of these, arsenic, given in large doses, is the best. I have repeatedly seen improvement under its use. On the other hand, there are curious remissions in the disease, as mentioned above, which render therapeutic deductions very fallacious.

Quinine may be given in cases with a malarial history. Iron may be of value in some cases, as may also inhalations of oxygen. Treatment with the X-rays should be tried. Some observers have reported very good results. Personally, I have not seen any very striking permanent improvement.

Excision of the leukæmic spleen has been performed 43 times, with 5 recoveries (J. C. Warren).

III. HODGKIN'S DISEASE

Definition.—A disease characterized by enlargement of the lymph-glands with progressive anæmia and a fatal termination.

Anatomically there is an increase in the adenoid tissue of the glands, proliferation of the endothelial cells, formation of mononuclear and multi-

and radium

but their duration is not yet determined. They contain
powerful explo

nuclear giant cells, the presence of eosinophiles, and thickening of the fibrous reticulum.

History.—In 1832 Hodgkin recorded a series of cases of enlargement of the lymphatic glands and spleen. From the motley group that Hodgkin described, Wilks picked out the disease and called it *anæmia lymphatica*. Other names that have been given to it are *adénie* by Trousseau, *pseudo-leukæmia* by Cohnheim, and *generalized lymphadenoma*.

Etiology.—A widely spread disease in Europe and America, a majority of the cases occur in young adults, and more frequently in males than in females. Twins and sisters have been known to be attacked. The cause is unknown. Certain features suggest an acute infection: the rapid course of some cases, the association with local irritation in the mouth and tonsils, the frequency with which the disease starts in the cervical glands, the gradual extension from one gland group to another, and the recurring exacerbations of fever. Various organisms have been described, but nothing definite has been determined. Possibly the disease is a spirillosis—in favor of which are the presence of eosinophilia, so characteristic of infection with animal parasites, the presence of eosinophilic cells in the glands, and the influence of arsenic on the disease. Sternberg suggested that the disease was a special form of tuberculosis; but the histological changes in the glands are quite characteristic, tubercle bacilli are not present in uncomplicated cases, the tuberculin test may be negative, and when present the tuberculosis appears to be a terminal infection.

Morbid Anatomy.—The superficial lymph glands are found most extensively involved, and from the cervical groups they form continuous chains uniting the mediastinal and axillary glands. The masses may pass beneath the pectoral muscles and even beneath the scapulæ. Of the internal glands, those of the thorax are most often affected, and the tracheal and bronchial groups may form large masses. The trachea and the aorta with its branches may be completely surrounded; the veins may be compressed, rarely the aorta itself. The masses perforate the sternum and invade the lung deeply. The retroperitoneal glands may form a continuous chain from the diaphragm to the inguinal canals. They may compress the ureters, the lumbar and sacral nerves, and the iliac veins. They may adhere to the broad ligament and the uterus and simulate fibroids. At an early stage the glands are soft and elastic; later they may become firm and hard. Fusion of contiguous glands does not often occur, and they tend to remain discrete, even after attaining a large size. The capsule may be infiltrated, and adjacent tissues invaded. On section the gland presents a grayish white semi-translucent appearance, broken by intersecting strands of fibrous tissue; there is no caseation or necrosis unless a secondary infection has occurred.

The spleen is enlarged in 75 per cent. of the cases; in young children the enlargement may be great, but the organ rarely reaches the size of the spleen in ordinary leukæmia. In more than half of the cases lymphoid growths are present.

The marrow of the long bones may be converted into a rich lymphoid tissue. The lymphatic structures of the tonsillar ring and of the intestines may show marked hyperplasia. The liver is often enlarged, and may present scattered nodular tumors, which may also occur in the kidneys.

Histology.—The studies of Andrewes and of Dorothy Reed show a very characteristic microscopic picture—proliferation of the endothelial and reticular cells, with the formation of lymphoid cells of uniform size and shape, and characteristic giant cells, the so-called lymphadenoma cells, containing four or more nuclei. Eosinophiles are always present, and proliferation of the stroma leads to fibrosis of the gland. The difference between the soft and hard forms depends largely upon the stage of the disease. When tuberculosis occurs as a secondary infection the two processes may be readily distinguished in sections of the gland.

Symptoms.—A tonsillitis may precede the onset. Enlargement of the cervical glands is usually an initial symptom; it is rare to find other superficial groups or the deeper glands attacked first. Months or even several years may elapse before the glands in the axillæ and groin become involved. During what may be called the first stage the patient's general condition is good; then anæmia comes on, not marked at first, but usually progressive. In the majority of cases the spleen is enlarged, but it never reaches the dimension of the leukæmic organ. There may be very little pain until the internal glands become involved. With swelling of the mediastinal glands there are cough, dyspnœa, and often intense cyanosis, with all the signs of intrathoracic tumor. There may be moderate fever. Bronzing of the skin may occur, apart altogether from the use of arsenic. Pruritus may be a very depressing symptom, and boils and ecthymatous blebs may occur. The leucocytes show no characteristic changes. There may be a moderate eosinophilia and, as the anæmia progresses, nucleated red corpuscles appear, and toward the end there are instances of a great increase in the lymphocytes. As the disease progresses there is marked emaciation with great asthenia, and sometimes anasarca. This represents the common clinical course, but there are many variations, among which the following are the most common:

(*a*) An ACUTE FORM has been described. I saw a remarkable case beginning, like so many cases of lymphatic leukæmia, with angina, in which the whole course was less than ten weeks. Ziegler mentions two cases of death within a month.

(*b*) LOCALIZED FORM.—The enlargement may be localized to certain groups, those in the neck, the groin, the retroperitoneum, or the thorax. Some of these cases present great difficulty in diagnosis, particularly when there are febrile paroxysms with very slight involvement of the external groups. The disease may be confined to one region for a year or more before there is any extension. The localized mediastinal group often presents a very remarkable picture—pressure signs, pain, orthopnœa—and, unless there are other groups involved, or enlargement of the spleen, it may be impossible to make the diagnosis during life.

(*c*) WITH RELAPSING PYREXIA.—To this remarkable type Pel and afterward Ebstein called attention. MacNalty has recently made a very careful study of this syndrome, which is one of the most remarkable met with in the practice of medicine. The relapsing pyrexia may occur in cases with involvement of the internal glands alone, or, more frequently, with a general involvement of all the groups. "Following on a period of low pyrexia, or of normal or subnormal temperature, there is a steady rise occupying two or four days to a maximum, which may reach 105°. For about three days the

Skin lesions. Lymphogranulomatosis.

H.V. Cole. J. clin. exp. 1927

many skin lesions - pruritus, prurigo-like exanthem - bronzelike p.s.
petechiae - urticaria. oedema.

2 groups. ① true lymphogranulomatosis - rare
(2) group of lesions

In rare cases breakdown. Ulc. like syphilitic lesion

temperature remains at a high level, and then there is a gradual fall by lysis occupying about three days, and the temperature then becomes sub-normal" (MacNalty). An afebrile period of ten days or two weeks then occurs, to be followed by another bout of fever. This may be repeated for many months. In one ~~of my~~ cases the pyrexia lasted for ~~accurately~~ fourteen days for many successive paroxysms. During the fever the glands swell and may become hot and tender. This febrile type may occur in connection with involvement of the internal glands alone. In one patient whose cervical glands had been thoroughly removed there were typical Pel-Ebstein paroxysms, and we could find no enlarged glands, internal or external.

(*d*) LATENT TYPE.—In his ~~recent~~ monograph Ziegler ~~has~~ called attention to the importance of this form, in which anæmia, fever, and constitutional symptoms may be present with enlargement of the internal glands. One of ~~my early~~ cases was of this type—a very stout man, in whom the retroperitoneal glands alone were involved. Symmers has reported an instance in which the glands and the hilus of the liver were attacked.

(*e*) SPLENOMEGALIC FORM.—Enlargement of the spleen is present in a large proportion of cases of Hodgkin's disease. Whether or not there is a type involving the spleen alone without the lymph glands is still a question. Formerly, under the name pseudo-leukæmia of Cohnheim, many cases of simple enlargement of the spleen with or without anæmia were spoken of as *pseudo-leukæmia splenica*. It is not improbable that the disease may originate in the lymphoid tissue of the spleen, and several cases have been reported ~~of late years~~ by Ziegler, Symmers, Warrington, and others. It must be very difficult to distinguish such cases clinically from the early stages of Banti's disease.

(*f*) Lastly, a LYMPHADENIA OSSIUM has been described—cases in which there have been multiple bone tumors of the bone marrow and of the periosteum with enlargement of the glands and spleen. How far these should be grouped with Hodgkin's disease seems to me very doubtful.

Diagnosis.—(*a*) TUBERCULOSIS.—There are both acute and chronic forms of general tuberculous adenitis (already described), but they do not often present difficulty in diagnosis. In the case of enlargement of the glands on one side of the neck beginning in a young person, it is often not at all easy to determine whether the disease is tuberculosis or beginning Hodgkin's disease. Two points should be decided. First, ~~under cocaine~~ one of the ~~small~~ glands of the affected ~~side~~ should be excised and the structure carefully studied. The histological changes differ markedly in Hodgkin's disease from those in tuberculosis. Secondly, tuberculin should be used if the patient is afebrile. In early tuberculosis of the glands of the neck the reaction is prompt and decisive. In the later stages, when many groups of glands are involved and the cachexia is well advanced, the tuberculin reaction may be present in Hodgkin's disease, but even then the histological changes are distinctive. Other points to be noted are the tendency in the tuberculous adenitis to coalescence of the glands, adhesion to the skin, with suppuration, etc., and the liability to tuberculosis of the lung or pleura.

(*b*) LEUKÆMIA.—As a rule, the blood examination gives the diagnosis at a glance, as Hodgkin's disease presents only a slight leucocytosis. A difficulty arises only in those rare instances of leukæmia, usually the acute lym-

phatic form, in which the leucocytes gradually decrease or in which the number for a time may become normal. Histologically there are striking differences between the structure of the glands in the two conditions.

(*c*) Lympho-sarcoma.—Clinically the cases may resemble Hodgkin's disease very closely, and in the literature the two diseases have been confounded. The glands, as a rule, form larger masses, the capsules are involved, and adjacent structures are attacked, but this may be the case in Hodgkin's disease. Pressure signs in the chest and abdomen are much more common in lympho-sarcoma. But the easiest and most satisfactory mode of diagnosis is examination of sections of a gland, as the structure is very different from that seen in Hodgkin's disease. The blood condition, the type of fever, etc., need a more careful study in this group of cases.

Course.—There are acute cases in which the enlargements spread rapidly and death follows in a few months. As a rule, the disease lasts for two or three years. Remarkable periods of quiescence may occur, in which the glands diminish in size, the fever disappears, and the general condition improves. Even a large group of glands may almost completely disappear, or a tumor mass on one side of the neck may subside while the inguinal glands are enlarging. Usually a cachexia with anæmia and swelling of the feet precedes death. A fatal event may occur early from great enlargement of the mediastinal glands.

Treatment.—When the glands are small and limited to one side of the neck, operation should be advised; even when both sides of the neck are involved, if there are no signs of mediastinal growth, operation is justifiable. The course of the disease may be delayed, even if cure does not follow.

There is a possibility that the X-rays may do good in selected cases. Certainly the glands have been reduced in size, but I know of no case in which complete cure has been reported. Local treatment of the glands seems to do but little good.

Arsenic is the only drug which has a positive value in the disease. In some cases the effects on the glands are striking. It may be given in the form of Fowler's solution in increasing doses. Recoveries have been reported (?). Ill effects from the larger doses are rare. Peripheral neuritis followed the use of ℥ iv, ʒj, ♏ xviij during a period of less than three months. Phosphorus is recommended by Gowers and Broadbent, and may be tried if arsenic is not well borne. Quinine, iron, and cod-liver oil are useful as tonics. For the pressure pains morphia should be given.

IV. PURPURA

Strictly speaking, purpura is a symptom, not a disease; but under this term are conveniently arranged a number of affections characterized by extravasations of the blood into the skin. In the present state of our knowledge a satisfactory classification can not be made. W. Koch groups all forms, including hæmophilia, under the designation *hæmorrhagic diathesis*, believing that intermediate forms link the mild purpura simplex and the most intense purpura hæmorrhagica. For a full discussion of the subject and an analysis of my cases, see Pratt's article in my "System of Medicine," Vol. IV.

The purpuric spots vary from 1 to 3 or 4 mm. in diameter. When small

Dresden

and pin-point-like they are called petechiæ; when large, they are known as ecchymoses. At first bright red in color, they become darker, and gradually fade to brownish stains. They do not disappear on pressure.

In some forms of purpura the coagulation time of the blood is retarded to ten or fifteen minutes, and in hæmophilia it has been delayed to fifty minutes.

The following is a provisional grouping of the cases:

Symptomatic Purpura.—(*a*) INFECTIOUS.—In pyæmia, septicæmia, and malignant endocarditis (particularly in the last affection) ecchymoses may be very abundant. In typhus fever the rash is always purpuric. Measles, scarlet fever, and more particularly small-pox and cerebro-spinal fever, have each a variety characterized by an extensive purpuric rash.

(*b*) TOXIC.—The virus of snakes produces extravasation of blood with great rapidity—a condition which has been very carefully studied by Weir Mitchell. Certain medicines, particularly copaiba, quinine, belladonna, mercury, ergot, and the iodides occasionally, are followed by a petechial rash. Purpura may follow the use of comparatively small doses of iodide of potassium. A fatal event may be caused by a small amount, as in a case reported by Stephen Mackenzie of a child which died after a dose of 2½ grains. An erythema may precede the hæmorrhage. It is not always a simple purpura, but may be an acute febrile eruption of great intensity. Workers with benzol, which is used as a solvent for rubber, may be attacked with severe purpura. Cases such as those reported by Selling have been in connection with the coating of tin cans, while the Swedish cases occurred in connection with the manufacture of bicycle tires. Under this division, too, comes the purpura so often associated with jaundice.

(*c*) CACHECTIC.—Under this heading are best described the instances of purpura which occur in the constitutional disturbance of cancer, tuberculosis, Hodgkin's disease, Bright's disease, scurvy, and in the debility of old age. In these cases the spots are usually confined to the extremities. They may be very abundant on the lower limbs and about the wrists and hands. This constitutes, probably, the commonest variety of the disease, and many examples of it can be seen in the wards of any large hospital.

(*d*) NEUROTIC.—One variety is met with in cases of organic disease. It is the so-called myelopathic purpura, which is seen occasionally in locomotor ataxia, particularly following attacks of the lightning pains and, as a rule, involving the area of the skin in which the pains have been most intense. Cases have been met with also in acute myelitis and in transverse myelitis, and occasionally in severe neuralgia. Another form is the remarkable hysterical condition in which stigmata, or bleeding points, appear upon the skin.

(*e*) MECHANICAL.—This variety is most frequently seen in venous stasis of any form, as in the paroxysms of whooping cough, and in epilepsy and about tight bandages.

Arthritic Purpura.—This form is characterized by involvement of the joints. It is usually known, therefore, as "rheumatic," though in reality the evidence upon which this view is based is not conclusive. Of 200 cases of purpura analyzed by Stephen Mackenzie, 61 had a history of rheumatism. For the present it seems more satisfactory to use the designation arthritic. Three groups of cases may be recognized:

(*a*) Purpura Simplex.—A mild form, often known as *purpura simplex,* seen most commonly in children, in whom, with or without articular pain, a crop of purpuric spots appears upon the legs, less commonly upon the trunk and arms. As pointed out by Graves, this form is not infrequently associated with diarrhœa. The disease is seldom severe. There may be loss of appetite, and slight anæmia. Fever is not, as a rule, present, and the patients get well in a week or ten days. Usually regarded as rheumatic, and certainly associated, in some instances, with undoubted rheumatic manifestations, yet in a majority of the patients the arthritis is slighter than in the ordinary rheumatism of children, and no other manifestations are present.

(*b*) Purpura (Peliosis) Rheumatica (*Schönlein's Disease*).—This remarkable affection is characterized by multiple arthritis and an eruption which varies greatly in character, sometimes *purpuric,* more commonly associated with *urticaria* or with *erythema exudativum.* The disease is most common in males between the ages of twenty and thirty. It not infrequently sets in with sore throat, a fever from 101° to 103°, and articular pains. The rash, which makes its appearance first on the legs or about the affected joints, may be a simple purpura or may show ordinary urticarial wheals. In other instances there are nodular infiltrations, not to be distinguished from erythema nodosum. The combination of wheals and purpura, the *purpura urticans,* is very distinctive. Much more rarely vesication is met with, the so-called *pemphigoid purpura.* The amount of œdema is variable; occasionally it is excessive. These are the cases which have been described as *febrile purpuric œdema.* The temperature range, in mild cases, is not high, but may reach 102° or 103° F.

The urine is sometimes reduced in amount and may be albuminous. The joint affections are usually slight, though associated with much pain, particularly as the rash comes out. Relapses may occur and the disease may return at the same time for several years in succession.

The diagnosis of Schönlein's disease offers no difficulty. The association of multiple arthritis with purpura and urticaria is very characteristic.

Schönlein's peliosis is thought by most writers to be of "rheumatic" origin, and certainly many of the cases have the characters of ordinary rheumatic fever, *plus* purpura. By many, however, it is regarded as a special affection, of which the arthritis is a manifestation analogous to that which occurs in hæmophilia and in scurvy. The frequency with which sore throat precedes the attack, and the occasional occurrence of endocarditis or pericarditis, are certainly very suggestive of true rheumatism.

The cases usually do well, and a fatal event is extremely rare. The throat symptoms may persist and give trouble. In two instances I have seen necrosis and sloughing of a portion of the uvula.

Visceral Lesions in Purpura.—In any form of purpura, in the erythemas, and in urticaria visceral lesions may occur. (*a*) Gastro-intestinal crises, pain, vomiting, melæna, and diarrhœa. The attacks have often been mistaken for appendicitis or for intussusception, and at operation the condition has been found to be an acute sero-hæmorrhagic infiltration of a limited area of the stomach or bowel. Identical attacks occur in angio-neurotic œdema. These crises may occur for years in children before an outbreak of purpura or urticaria gives a clue to their nature. (*b*) Enlargement of the

Tirer au chevalet.

spleen is usually present in these cases. (*c*) Albuminuria and acute nephritis may occur and form the most serious complication, of which seven cases in my series died (*Am. J. Med. Sciences,* Jan., 1904). The combination of purpura with colic is usually spoken of as Henoch's purpura.

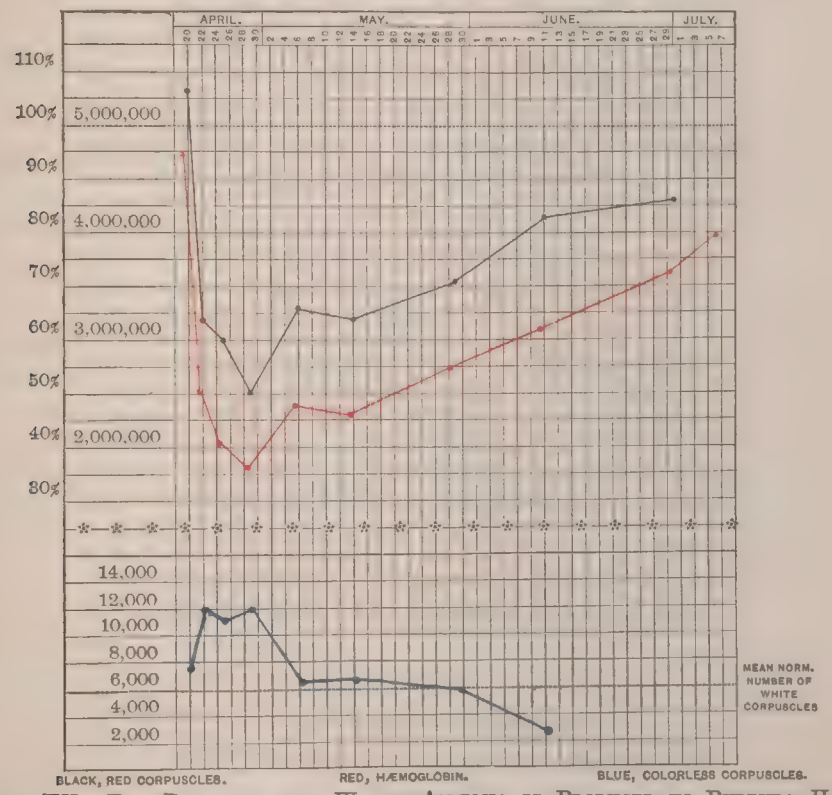

CHART XX.—THE RAPIDITY WITH WHICH ANÆMIA IS PRODUCED IN PURPURA HÆMORRHAGICA, AND THE GRADUAL RECOVERY.

Purpura Hæmorrhagica.—Under this heading may be considered cases of very severe purpura with hæmorrhages from the mucous membranes. The affection, known as the *morbus maculosus* of Werlhof, is most commonly met with in young and delicate individuals, particularly in girls; but the disease may attack adults in full vigor. After a few days of weakness and debility, purpuric spots appear on the skin and rapidly increase in number and size. Bleeding from the mucous surfaces sets in, and the epistaxis, hæmaturia, and hæmoptysis may cause profound anæmia. Death may take place from loss of blood, or from hæmorrhage into the brain. Slight fever usually accompanies the disease. In favorable cases the affection terminates in from ten days to two weeks. There are instances of purpura hæmorrhagica of great malignancy, which may prove fatal within twenty-four hours—*purpura fulminans.* This form is most commonly met with in children, and is characterized chiefly by cutaneous hæmorrhages, and death may occur before any bleeding takes place from the mucous membranes.

In the *diagnosis* of purpura hæmorrhagica it is important to exclude scurvy, which may be done by the consideration of the previous health, the circumstances under which the disease occurs, and by the absence of swelling of the gums. The malignant forms of the fevers, particularly small-pox and measles, are distinguished by the prodromes and the higher temperature.

Treatment.—In symptomatic purpura attention should be paid to the conditions under which it occurs, and measures should be employed to increase the strength and to restore a normal blood condition. Tonics, good food, and fresh air meet these indications. In the simple purpura of children, or that associated with slight articular trouble, arsenic in full doses should be given. No good is obtained from the small doses, but the Fowler's solution should be pushed freely until physiological effects are obtained. In peliosis rheumatica the sodium salicylate may be given, but with discretion. I confess not to have seen any special control of the hæmorrhages by this remedy.

Aromatic sulphuric acid, ergot, turpentine, acetate of lead, or tannic and gallic acids may be given, and in some instances they seem to check the bleeding. Oil of turpentine is perhaps the best remedy, in 10 or 15-minim (1 c. c.) doses three or four times a day. The calcium salts, preferably the lactate, may be given in doses of 15 grains (1 gm.) three or four times a day for a few days, to increase the coagulability of the blood. In bleeding from the mouth, gums, and nose the inhalation of carbon dioxide, irrigations with 2-per-cent. gelatin solution, and adrenalin should be tried. The last remedy has often acted promptly. The treatment of the severe forms is the same as that given in hæmophilia.

HÆMORRHAGIC DISEASES OF THE NEW-BORN

Syphilis Hæmorrhagica Neonatorum.—The child may be born healthy, or there may be signs of hæmorrhage at birth. Then in a few days there are extensive cutaneous extravasations and bleeding from the mucous surfaces and from the navel. The child may become deeply jaundiced. The post mortem shows numerous extravasations in the internal organs and extensive syphilitic changes in the liver and other organs.

Epidemic Hæmoglobinuria (*Winckel's Disease*).—Hæmoglobinuria in the new-born, which occasionally occurs in epidemic form in lying-in institutions, is a very fatal affection, which sets in usually about the fourth day after birth. The child becomes jaundiced, and there are marked gastro-intestinal symptoms, with fever, jaundice, rapid respiration, and sometimes cyanosis. The urine contains albumin and blood coloring matter—methæmoglobin. The disease has to be distinguished from the simple icterus neonatorum, with which there may sometimes be blood or blood coloring matter in the urine. The post mortem shows an absence of any septic condition of the umbilical vessels, but the spleen is swollen, and there are punctiform hæmorrhages in different parts. Some cases have shown in a marked degree acute fatty degeneration of the internal organs—the so-called Buhl's disease.

Morbus Maculosus Neonatorum.—Apart from the common visceral hæmorrhages, the result of injuries at birth, bleeding from one or more of the surfaces is a not uncommon event in the new-born, particularly in hospital practice. Forty-five cases occurred in 6,700 deliveries (C. W. Townsend).

Blood platelets are decreased, there is delayed bleeding time &c
non-retractile soft blood clot.

Diag. other purpuras platelets are normal = Diag. from non-leukaemic
atypes of leukaemia.

The existence of blood serum is still effected &c.
coagulens?

The bleeding may be from the navel alone, but more commonly it is general. Of Townsend's 50 cases, in 20 the blood came from the bowels (*melæna neonatorum*), in 14 from the stomach, in 14 from the mouth, in 12 from the nose, in 18 from the navel, in 3 from the navel alone. The bleeding begins within the first week, but in rare instances is delayed to the second or third. Thirty-one of the cases died and 19 recovered. The disease is usually of brief duration, death occurring in from one to seven days. The temperature is often elevated. The nature of the disease is unknown. As a rule, nothing abnormal is found post mortem. The general and not local nature of the affection, its self limited character, the presence of fever, and the greater prevalence of the disease in hospitals suggest an infectious origin (Townsend). The bleeding may be associated with intense hæmatogenous jaundice. Not every case of bleeding from the stomach or bowels belongs in this category. Ulcers of the œsophagus, stomach, and duodenum have been found in the new-born dead of *melæna neonatorum*. The child may draw the blood from the breast and subsequently vomit it.

V. HÆMOPHILIA

Definition.—A disease characterized by excessive and chronic liability to immoderate hæmorrhage. The liability is hereditary, and is confined to the male sex (Bulloch and Fildes).

History.—The origin of our knowledge of this remarkable condition dates from 1803, when John C. Otto, a Philadelphia physician, published "an account of an hæmorrhagic disposition occurring in certain families"; and he first used the word "bleeder." The works of Grandidier and of Wickham Legg give full clinical details, and the monograph of Bulloch and Fildes (Dulan & Co., London, 1911) presents in extraordinary detail every aspect of the disease.

Distribution.—A majority of the cases have been reported from Germany, Switzerland, and the United States. Jews are supposed to be more prone than others to the disease, but this Bulloch doubts, and he discredits the negro cases.

Sex.—Bulloch and Fildes claim to have established by their researches the fact of immunity in females, denying the authenticity of all the published cases (19). "In none of the families of bleeders . . . do we find any unequivocal evidence of abnormality in the women, that is to say, any abnormality beyond what might be expected in any collection of females taken at random."

Inheritance.—Otto pointed out in his original paper that while the females do not themselves bleed they alone transmit the tendency. Of 171 recorded instances of transmission, 160 conform to the "law of Nasse" that the disease is transmitted by the unaffected female—"the conductor" (Bulloch and Fildes). They explain the 11 exceptions, and conclude that the disease is not capable of being propagated through a male. Instances of hæmophilia without demonstrable inheritance are very rare.

Pathology.—Recent studies point to disturbance in the fibrin forming factors as the essential feature of the disease. Almroth Wright showed that the coagulation time in hæmophilics was much delayed, and the recent ob-

servations of Addis in 12 cases have shown that the delay may be as much as 40 to 60 minutes. According to the development of Buchanan and Schmidt's views the factors in coagulation are fibrinogen, prothrombin, thrombokinase, and calcium, and by the interaction of the last three thrombin is produced, which, acting on the fibrinogen, precipitates fibrin. Set free when a wound takes place, the thrombokinase present in the tissues in the presence of calcium rapidly forms a new body—thrombin. Sahli, whose studies on hæmophilia have been most important and interesting, believes that there is an anomaly of the cellular elements of the blood, and of certain other cells, especially in the walls of the vessels, so that the thrombokinase is not produced. Morowitz believes that thrombokinase is derived from the leucocytes or from the platelets. One of the difficulties in explaining the bleeding in hæmophilia is the fact that the hæmorrhage continues in spite of the presence of clots in and about the wound. Addis believes that a higher amount of thrombokinase is required to produce rapid clotting in hæmophilic than in normal blood. In a wound, coagulation may occur only in those parts, as at the side, where the concentration of this material is highest; but the clot itself prevents the addition of further quantities of the thrombokinase from the tissues, and when the quantity of thrombin set free from the primary clot is insufficient completely to coagulate the blood in the centre of the wound, the bleeding may continue indefinitely.

Symptoms.—"The cardinal symptoms are three in number . . . an *inherited* tendency in *males* to *bleed*" (Bulloch and Fildes). A trifling injury, of no moment in a normal person, determines a hæmorrhage, which has no tendency to stop, but the blood trickles or oozes until death follows or there is spontaneous arrest. The bleeding may be external, internal, or into joints. A majority of the attacks may be traced to trauma but spontaneous bleeding may occur. The liability is first noticed in children and persists to adult life, gradually diminishing and eventually disappearing. Tooth extraction is a very common cause. Epistaxis is a frequent occurrence, heading the list in Grandidier's series of 334 cases. Other localities were: mouth 43, stomach 15, bowels 36, urethra 16, lungs 17, and a few instances of bleeding from the tongue, finger-tips, tear papilla, eyelids, external ear, vulva, navel, and scrotum. Trivial operations, as circumcision, have been followed by fatal hæmorrhage.

Hæmarthrosis and periarticular bleedings are common. The knee is most commonly attacked, and the affection has been mistaken for tuberculosis. König distinguishes three stages—hæmarthrosis, panarthritis, and deformity.

Diagnosis.—Karl Pearson's new iatro-mathematical school of medicine has done good work in making the profession more careful about its facts, as well as its figures. Bulloch's monograph should be read by all who value accuracy of observation and of investigation. Forms of bleeding are so common that it is a simple matter to construct a pedigree showing an inherited "hæmorrhagic diathesis." It is essential for the diagnosis that the individual should have been more or less subject to bleeding from various parts throughout his life. "No solitary hæmorrhage, however inexplicable, should, in our opinion, be regarded as hæmophilia; it is necessary to show that the individual has been repeatedly attacked, if not from birth, from infancy" (Bulloch and Fildes).

In the diagnosis from purpura the following points are important.
Purpura of the skin rarely causes hemorrhage in h., in p. it is the rule. The application of a tourniquet to the upper arm is negative in h, in purpura it usually is petechiae in the forearm. The platelets are normal in h, much decreased in purpura. There is delay in the coag. time in haem.; it is normal or almost so in h. The "bleeding time" is not increased in h. it is increased in purpura.
Blood clot in h. retracts normally, not in p.

Injection v 5% Sol. Optochin hydrochlor. carbamat ?!

Treatment.—Recent work of a most encouraging character indicates that injections of serum or of defibrinated blood, or direct transfusion have remarkable effects in these severe hæmorrhagic cases, particularly in hæmophilia. The method has been introduced by Weil, who recommends the subcutaneous injection of 30 c. c. of fresh human or animal serum or the intravenous injection of 15 c. c. The method has been successful in the hæmorrhagic diseases of the new-born, injecting subcutaneously 10 c. c., and giving as much as 100 or even 200 c. c. in four or five days (Moss). The serum may be applied locally to the bleeding spot in hæmophilia. Cure has followed the use of the anti-diphtheritic serum. Injection of defibrinated blood has been successful in several cases, and melæna neonatorum has been cured by direct transfusion. Lambert's case is remarkable, as the hæmorrhage had been going on for three days and the child was dying. In the severer cases the direct transfusion should be tried, and the technique is not now a matter of difficulty.

VI. ERYTHRÆMIA

(Vaquez' Disease, Polycythæmia Vera)

Definition.—A disease characterized by a persistent increase of the red blood-corpuscles, a condition of plethora, splenomegaly, and at times cyanosis. Cases were reported by Rendu and Widal, Vaquez, Cabot, McKeen, Saundby and Russell, and since 1903, when I tried to put the disease on a firm clinical basis, great interest has been aroused in the condition.

Pathology.—We see polycythæmia as a secondary condition in high altitudes, and in stasis of the blood in congenital heart disease, and in emphysema of the lungs. The high altitude hyperglobulism is compensatory to lack of oxygen in the air, and there is an increased activity of the bone marrow. In erythræmia proper this same increased activity of the bone marrow is present, and the disease is regarded as a primary lesion of the erythroblastic tissues of the bone marrow, just as leukæmia is an affection of the leucoblastic elements. There is also an increased viscosity of the blood which favors the stasis and capillary engorgement.

Symptoms.—The three cardinal features are a change in the appearance of the patient, enlargement of the spleen, and hyperglobulism. The superficial blood vessels, capillaries, and veins look full, so that the skin is always congested, in warm weather of a brick red color, in cold weather cyanosed. The engorgement of the face may be extreme, extending to the conjunctivæ, and in the cold the cyanosis of the face and hands may be as marked as any that is ever seen. There is often, too, a remarkable vasomotor instability, e. g., the hand becoming deeply engorged when held down, and rapidly anæmic when held up.

The spleen is usually enlarged, but not to the great extent of leukæmia. It may vary in size from time to time. It is hard, firm, and painless.

The total bulk of blood is enormously increased, and the ratio of corpuscles to plasma is high. The polycythæmia ranges from 7 to 12 or even 13 millions of red corpuscles per c. mm. As a rule, they are normal in appearance and shape; nucleated red blood-corpuscles may be present, the hæ-

moglobin ranging from 130 to 160 per cent., but the color index is relatively low. Moderate leucocytosis is the rule without any characteristic differential change; a few myelocytes may be present. The specific gravity is high.

Of other symptoms the most common are headache, flushing, and giddiness. Constipation is common, and albuminuria is usually present. The blood pressure is high; occasionally there may be hæmorrhages into the skin and from the mucous membranes. Recurring ascites, probably in association with splenic tumor, was present in two of my cases.

Morris has reported a couple of cases with the general appearance of the disease and with slight enlargement of the spleen, but without polycythæmia. Geisböck has described a variety, *polycythæmia hypertonica*, with increased tension, arterio-sclerosis, and nephritis.

Diagnosis.—The triad of features above referred to are sufficient in the absence of congenital heart disease, emphysema, and forms of cyanosis associated with poisoning by coal tar products. In a few rare cases the polycythæmia has been associated with tuberculosis of the spleen.

Prognosis.—The prognosis is bad for cure, but the condition may persist for years with reasonably good health. Cardiac failure, hæmorrhage, and recurring ascites have been the usual modes of death.

Treatment.—When there is much fullness of the head and vertigo, repeated bleedings have given relief. Inhalations of oxygen may be tried when the cyanosis is extreme. Saline purges and low diet are also helpful. The X-rays have done no good in my cases. Splenectomy should not be performed.

VII. ENTEROGENOUS CYANOSIS

(*Methæmoglobinæmia and Sulphæmoglobinæmia*)

Definition.—A form of permanent cyanosis due to changes in the composition of the hæmoglobin of the blood.

Etiology.—It has long been known that with the use of certain drugs changes were induced in the hæmoglobin. In poisoning by potassium chlorate methæmoglobinæmia occurs often with an active hæmolysis. Carbon monoxide, sulphuretted hydrogen, the coal-tar products, acetanilide, phenacetin, sulphonal, and trional may cause a chronic cyanosis. Stokvis brought forward evidence to show that certain cases of chronic cyanosis are associated with intestinal disturbances, and he gives this form the name "enterogenous." Some of the forms are associated with methæmoglobinæmia, others with sulphæmoglobinæmia. In a doubtful case, with absence of lesions of the heart or lungs, a spectroscopic examination of the blood will determine if the cyanosis is of this nature, and which of the two derivatives of hæmoglobin is causing it.

There have been some 15 or 20 cases now on record of both forms (see Garrod, Allbutt and Rolleston's System).

Methæmoglobinæmia.—Several of the patients have had chronic diarrhœa, in two associated with parasites. In Stokvis' case there was clubbing of the fingers without any recognizable cause. Gibson and Douglas obtained from the blood of their patient a pure culture of a colon organism and suggested

some have found a high % of mononuclears

in capillary fingerwork.

food poor in purines + rich in meat

Benzol 3 qm. a day has helped.

Jenner quoted by the blind count.

Mitchell gave up Sunday

began with 1 qm t.i.d.

Benzol 1

Oliv. oil 1

Methyl pyramin 2.

the name "Microbic cyanosis." In connection with this observation it may be mentioned that methæmoglobinæmia has been met with in Winckel's disease, in one case of which the staphylococcus has been isolated from the blood. But a still more striking confirmation is Boycott's discovery of an infective methæmoglobinæmia in rats, caused by Gaertner's bacillus, which gives a remarkable bluish tint to the skin of white rats.

Sulphæmoglobinæmia.—The appearance of the patients is very much the same. They look very badly, even death-like, but feel comfortable, and there is no shortness of breath. The absence of symptoms at once suggests a drug habit as a cause, but the history is negative. In the case reported by Wood Clarke, the first in this country, the cultures were negative. Intestinal disturbances have been present in a number of cases, and Garrod suggests that it is a chronic poisoning by hydrogen sulphide, possibly absorbed from the intestines.

SECTION IX

DISEASES OF THE CIRCULATORY SYSTEM

A. DISEASES OF THE PERICARDIUM

I. PERICARDITIS

Pericarditis is the result of infective processes, primary or secondary, or arises by extension of inflammation from contiguous organs.

Etiology.—PRIMARY, so-called idiopathic, inflammation is rare; but it has been met with in children without any evidence of rheumatism or of any local or general disease. Certain of the cases are tuberculous.

Pericarditis from injury usually comes under the care of the surgeon in connection with the primary wound. The trauma may be from within, due to the passage of a foreign body—a needle, a pin, or a bone—through the œsophagus—a variety exceedingly common in cows and horses.

SECONDARY: (*a*) Occurs most frequently in connection with rheumatic fever. The percentage given by different authors ranges from thirty to seventy. In our 330 cases of rheumatic fever (Johns Hopkins Hospital) pericarditis occurred in twenty—practically 6 per cent. The articular trouble may be slight or, indeed, the disease may be associated with acute tonsillitis in rheumatic subjects. Certain of the so-called idiopathic cases have their origin in an acute tonsillitis. The pericarditis may precede the arthritis. (*b*) In septic processes; in the acute necrosis of bone and in puerperal fever it is not uncommon. (*c*) In tuberculosis, in which the disease may be primary or part of a general involvement of the serous sacs or associated with extensive pulmonary disease. (*d*) In the fevers. Not infrequent after scarlatina, it is rare in measles, small-pox, typhoid fever, and diphtheria. In pneumonia it is not uncommon, occurring in 31 among 665 in my clinic (J. A. Chatard). In 184 post mortems there were 29 instances of pericarditis. It is most frequent in double pneumonia, and in our series with disease of the right side, if only one lung was involved. Pericarditis sometimes complicates chorea; it was present in 19 of 73 autopsies which I collected; in only 8 of these was arthritis present. (*e*) Terminal pericarditis. In gout, in chronic Bright's disease —*pericardite brightique* of the French—in arterio-sclerosis, in scurvy, in diabetes, and in chronic illness of all sorts a latent pericarditis is common and is usually overlooked.

(*f*) *By Extension.*—In pleuro-pneumonia it forms a serious complication, and was present in 5 cases of 100 post mortems. It is most often met with in the pleuro-pneumonia of children and of alcoholics. With simple pleurisy it is rare. In ulcerative endocarditis, purulent myocarditis, and in aneurism

of the aorta pericarditis is occasionally found. It may also follow extension of the disease from the mediastinal glands, the ribs, sternum, vertebræ, and even from the abdominal viscera.

The ordinary pus cocci, the pneumococcus, and the tubercle bacillus are the chief organisms met with in acute pericarditis.

Pericarditis occurs at all ages. Cases have been reported in the fetus. In the new-born it may result from septic infection through the navel. Throughout childhood the incidence of rheumatic fever and scarlet fever makes it a frequent affection, whereas late in life it is most often associated with tuberculosis, ~~Bright's disease~~, and gout. Males are somewhat more neph. frequently attacked than females. The so-called epidemics of pericarditis have been outbreaks of pneumonia with this as a frequent complication.

ACUTE FIBRINOUS PERICARDITIS

This, the most common and benign form, is distinguished by the small amount of exudate which coats the surface in a thin layer.

It may be partial or general. In the mildest grades the membrane looks lustreless and roughened, due to the presence of a thin fibrinous sheeting, which can be lifted with the knife, showing beneath an injected or ecchymotic serosa. As the fibrinous sheeting increases in thickness the constant movement of the adjacent surfaces gives to it sometimes a ridge-like, at others a honeycombed appearance. With more abundant fibrinous exudation the membranes present an appearance resembling buttered surfaces which have been drawn apart. The fibrin is in long shreds, and the heart presents a curiously shaggy appearance—the hairy heart of old writers, *cor villosum*.

In mild grades the subjacent muscle looks normal, but in the more prolonged and severe cases there is myocarditis, and for 2 or 3 mm. beneath the visceral layer the muscle presents a pale, turbid appearance. Many of these acute cases are tuberculous and the granulations are easily overlooked in a superficial examination.

There is usually a slight amount of fluid entangled in the meshes of fibrin, but there may be very thick exudate without much serous effusion.

Symptoms.—Unless sought for there may be no objective signs, and for this reason it is often overlooked, and in hospitals the disease is relatively more common in the post mortem room than in the wards.

Pain is a variable symptom, not usually intense, and in this form rarely excited by pressure. It is more marked in the early stage, and may be referred either to the præcordia or to the region of the xiphoid cartilage. Instances are recorded of pain of an aggravated and most distressing character resembling angina. Fever is usually present, but it is not always easy to say how much depends upon the primary disease, and how much upon the pericarditis. It is as a rule not high, rarely exceeding 102.5° F. In rheumatic cases hyperpyrexia has been observed.

Physical Signs.—*Inspection* is negative; *palpation* may reveal the presence of a distinct fremitus caused by the rubbing of the roughened pericardial surfaces. This is usually best marked over the right ventricle. It is not always to be felt, even when the friction sound on auscultation is loud and clear. *Auscultation:* The friction sound, due to the movement of the peri-

cardial surfaces upon each other, is one of the most distinctive of physical signs. It is double, corresponding to the systole and diastole; but the synchronism with the heart sounds is not accurate, and the to and fro murmur usually outlasts the time occupied by the first and second sound. In rare instances the friction is single; more frequently it appears to be triple in character—a sort of canter rhythm. The sounds have a peculiar rubbing, grating quality, characteristic when once recognized, and rarely simulated by endocardial murmurs. Sometimes instead of grating there is a creaking quality—the *bruit de cuir neuf*—the new leather murmur of the French. The pericardial friction appears superficial, very close to the ear, and is usually intensified by pressure with the stethoscope. It is best heard over the right ventricle, the part of the heart which is most closely in contact with the front of the chest—that is, in the fourth and fifth interspaces and adjacent portions of the sternum. There are instances in which the friction is most marked at the base, over the aorta, and at the superior reflection of the pericardium. Occasionally it is best heard at the apex. It may be limited to a very narrow area, or ~~it may be~~ transmitted up and down the sternum. There are, however, no definite lines of transmission as in endocardial murmurs. An important point is the variability of the sounds, both in position and quality; they may be heard at one visit and not at another. The maximum of intensity will be found to vary with position. Friction may be present with a thin, almost imperceptible, layer of exudate; on the other hand it may not be present with a thick, buttery layer. The rub may be entirely obscured by the loud bronchial râles in pneumonia, in which disease pericarditis is not recognized clinically in more than half the cases, only 13 in 31 cases in ~~my~~ series.

Diagnosis.—There is rarely any difficulty in determining the presence of a dry pericarditis, for the friction sounds are distinctive. The double murmur of aortic ~~incompetency~~ may simulate closely the to and fro pericardial rub. ~~I recall several instances in which this mistake was made.~~ The constant character of the aortic murmur, the direction of transmission, the phenomena in the arteries, and the associated conditions ~~of the disease~~ should ~~be sufficient to~~ prevent this error.

Pleuro-pericardial friction is very common, and may be associated with endo-pericarditis, particularly in cases of pleuro-pneumonia. It is frequent, too, in tuberculosis. It is best heard over the left border of the heart, and is much affected by the respiratory movement. Holding the breath or taking a deep inspiration may annihilate it. The rhythm is not the simple to and fro diastolic and systolic, but the respiratory rhythm is superadded, usually intensifying the murmur during expiration and lessening it on inspiration. In tuberculosis of the lungs there are instances in which, with the friction, a loud systolic click is heard, due to the compression of a thin layer of lung and the expulsion of a bubble of air from a small softening focus or from a bronchus.

And, lastly, it is not very uncommon, in the region of the apex beat, to hear a series of fine crepitant sounds, systolic in time, often very distinct, suggestive of pericardial adhesions, but heard too frequently for this cause.

Course and Termination.—Simple fibrinous pericarditis never kills, but it occurs so often in connection with serious affections that we have frequent

opportunities to see all stages of its progress. In the majority of cases the inflammation subsides and the thin fibrinous laminæ gradually become converted into connective tissue, which unites the pericardial leaves firmly together. A very thin layer may "clear" without leaving adhesions. In other instances the inflammation progresses, with increase of the exudation, and the condition is changed from a "dry" to a "moist" pericarditis, or the pericarditis with effusion.

In a few instances—probably always tuberculous—the simple plastic pericarditis becomes chronic, and great thickening of both visceral and parietal layers is gradually induced.

PERICARDITIS WITH EFFUSION

Etiology.—Commonly a direct sequence of the dry or plastic pericarditis, of which it is sometimes called the second stage, this form is found most frequently in association with rheumatic fever, tuberculosis, and septicæmia, and sets in usually with ~~the symptoms above described, namely,~~ præcordial pain, with slight fever or a distinct chill.

In children the disease may, like pleurisy, come on without local symptoms, and, after a week or two of failing health, slight fever, shortness of breath, and increasing pallor, the physician may find, to his astonishment, signs of ~~most~~ extensive pericardial effusion. These latent cases are often tuberculous. W. Ewart has called special attention to latent and ephemeral pericardial effusions, which he thinks are often of short duration and of moderate size, with an absence of the painful features of pericarditis.

Morbid Anatomy.—The effusion may be sero-fibrinous, hæmorrhagic, or purulent. The amount varies from 200 to 300 c. c. to 2 litres. In the cases of sero-fibrinous exudation the pericardial membranes are covered with thick, creamy fibrin, which may be in ridges or honeycombed, or may present long, villous extensions. The parietal layer may be several millimetres in thickness and ~~may~~ form a firm, leathery membrane. The hæmorrhagic exudation is usually associated with tuberculous, or ~~with~~ cancerous pericarditis, or with the disease in the aged. The lymph is less abundant, but both surfaces are injected and often show numerous hæmorrhages. Thick, curdy masses of lymph are usually found in the dependent part of the sac. In many cases the effusion is really sero-purulent, a thin, turbid exudation containing flocculi of fibrin.

The pericardial layers are greatly thickened and covered with fibrin. When the fluid is pus, they present a grayish, rough, granular surface. Sometimes there are distinct erosions on the visceral membrane. The heart muscle in these cases becomes involved to a greater or less extent and, on section, the tissue, for a depth of from 2 to 3 mm., is pale and turbid, and shows evidence of fatty and granular change. Endocarditis coexists frequently, but rarely results from the extension of the inflammation through the wall of the heart.

Symptoms.—Even with copious effusion the onset and course may be so insidious that no suspicion of the true nature of the disease is aroused.

As in the simple pericarditis, pain may be present, either sharp and stabbing or as a sense of distress and discomfort in the cardiac region. It is more frequent with effusion than in the plastic form. Pressure at the lower end of

the sternum usually aggravates it. Dyspnœa is a common and important symptom, one which, perhaps, more than any other, excites suspicion of grave disorder and leads to careful examination of heart and lungs. The patient is restless, lies upon the left side or, as the effusion increases, sits up in bed. Associated with the dyspnœa is in many cases a peculiarly dusky, anxious countenance. The pulse is rapid, small, sometimes irregular, and may present the characters known as *pulsus paradoxus*, in which during each inspiration the pulse beat becomes very weak or is lost. These symptoms are due, in great part, to the direct mechanical effect of the fluid within the pericardium which embarrasses the heart's action. Other pressure effects are distention of the veins of the neck, dysphagia, which may be a marked symptom, and irritative cough from compression of the trachea. Aphonia is not uncommon, owing to compression or irritation of the recurrent laryngeal as it winds round the aorta. In massive effusion the pericardial sac occupies a large portion of the antero-lateral region of the left side and the condition has frequently been mistaken for pleurisy. Even in moderate grades the left lung is somewhat compressed, an additional element in the production of the dyspnœa.

Great restlessness, insomnia, and in the later stages low delirium and coma are symptoms in the more severe cases. Delirium and marked cerebral symptoms are associated with the hyperpyrexia of rheumatic cases, but apart from the ordinary delirium there may be peculiar mental symptoms. The patient may become melancholic and show suicidal tendencies. In other cases the condition resembles closely delirium tremens. Sibson, who has specially described this condition, states that the majority of such cases recover. Chorea may also occur, as was pointed out by Bright. Epilepsy is a rare complication which has occurred during paracentesis.

Physical Signs.—*Inspection.*—In children the præcordia bulges and with copious exudation the antero-lateral region of the left chest becomes enlarged. A wavy impulse may be seen in the third and fourth interspaces, or there may be no impulse visible. The intercostal spaces bulge somewhat and there may be marked œdema of the wall. The epigastrium may be more prominent. Perforation externally through a space is very rare. Owing to the compression of the lung, the expansion of the left side is greatly diminished. The diaphragm and left lobe of the liver may be pushed down and may produce a distinct prominence in the epigastric region.

Palpation.—A gradual diminution and final obliteration of the cardiac impulse is a striking feature in progressive effusion. The position of the apex beat is not constant. In large effusions it is usually not felt. In children as the fluid collects the pulsation may be best seen in the fourth space, but this may not be the apex itself. The pericardial friction may lessen with the effusion, though it often persists at the base when no longer palpable over the right ventricle, or may be felt in the erect and not in the recumbent posture. Fluctuation can rarely, if ever, be detected.

Percussion gives most important indications. The gradual distention of the pericardial sac pushes aside the margins of the lungs so that a large area comes in contact with the chest wall and gives a greatly increased percussion dulness. The form of this dulness is irregularly pear-shaped; the base or broad surface directed downward and the stem or apex directed upward toward the manubrium. There is a disproportionate extension of dulness

upward and to the right, with dulness in the right fifth interspace extending one or two inches to the right of the sternum (Rotch's sign). The dulness may extend to the left beyond the apex beat. There may be marked differences in the area of flatness in the erect and recumbent postures. In large effusions there may be impaired resonance in the left axilla, and Bamberger called attention to an area of dulness near the angle of the scapula with bronchial breathing, which may alter when the patient leans forward.

Auscultation.—The friction sound heard in the early stages may disappear when the effusion is copious, but often persists at the base or at the limited area of the apex. It may be audible in the erect and not in the recumbent posture. With the absorption of the fluid the friction returns. One of the most important signs is the gradual weakening of the heart sounds, which with the increase in the effusion may become so muffled and indistinct as to be scarcely audible. The heart's action is usually increased and the rhythm disturbed. Occasionally a systolic endocardial murmur is heard. Early and persistent accentuation of the pulmonary second sound may be present.

Important accessory signs in large effusion are due to pressure on the left lung. The antero-lateral margin of the lower lobe is pushed aside and in some instances compressed, so that percussion in the axillary region, in and just below the transverse nipple line, gives a modified percussion note, usually a flat tympany. Variations in the position of the patient may change materially this modified percussion area, over which on auscultation there is either feeble or tubular breathing.

Course.—Cases vary extremely in the rapidity with which the effusion takes place. In every instance, when a pericardial friction murmur has been detected, the practitioner should first outline with care—using the aniline pencil—the upper and lateral limits of cardiac dulness, secondly mark the position of the apex beat, and thirdly note the intensity of the heart sounds. In many instances the exudation is slight in amount, reaches a maximum within forty-eight hours, and then gradually subsides. In other instances the accumulation is more gradual and progressive, increasing for several weeks. To such cases the term *chronic* has been applied. The rapidity with which a sero-fibrinous effusion may be absorbed is surprising. The possibility of the absorption of a purulent exudate is shown by the cases in which the pericardium contains semi-solid grayish masses in all stages of calcification. With sero-fibrinous effusion, if moderate in amount, recovery is the rule, with inevitable union, however, of the pericardial layers. In some of the septic cases there is a rapid formation of pus and a fatal result may follow in three or four days. More commonly, when death occurs with large effusion, it is not until the second or third week and takes place by gradual asthenia.

Prognosis.—In the sero-fibrinous effusions the outlook is good, and a large majority of all the rheumatic cases recover. The purulent effusions are, of course, more dangerous; the septic cases are usually fatal, and recovery is rare in the slow, insidious tuberculous forms.

Diagnosis.—Probably no serious disease is so frequently overlooked by the practitioner. Post mortem experience shows how often pericarditis is not recognized, or goes on to resolution and adhesion without attracting notice. In a case of rheumatic fever, watched from the outset, with the attention directed daily to the heart, it is one of the simplest of diseases to diagnose;

but when one is called to a case for the first time and finds perhaps an increased area of præcordial dulness, it is often very hard to determine with certainty whether or not effusion is present.

The difficulty usually lies in distinguishing between dilatation of the heart and pericardial effusion. Although the differential signs are simple enough on paper, it is notoriously difficult in certain cases, particularly in stout persons, to say which of the conditions exists. The points which deserve attention are:

(*a*) The character of the impulse, which in dilatation, particularly in thin-chested people, is commonly visible and wavy.

(*b*) The shock of the cardiac sounds is more distinctly palpable in dilatation.

(*c*) The area of dulness in dilatation rarely has a triangular form; nor does it, except in cases of mitral stenosis, reach so high along the left sternal margin or so low in the fifth and sixth interspaces *without visible or palpable impulse.* An upper limit of dulness shifting with change of position speaks strongly for effusion.

(*d*) In dilatation the heart sounds are clearer, often sharp, valvular, or fetal in character; gallop rhythm is common, whereas in effusion the sounds are distant and muffled.

(*e*) Rarely in dilatation is the distention sufficient to compress the lung and produce the tympanitic note in the axillary region, or flatness behind.

(*f*) The X-ray picture may be very definite, and unlike any form of dilatation or hypertrophy of the heart.

The number of excellent observers who have acknowledged that they have failed sometimes to discriminate between these two conditions, and who have indeed performed paracentesis *cordis* instead of paracentesis *pericardii,* is perhaps the best comment on the difficulties.

Massive (1½ to 2-litre) exudations have been confounded with a pleural effusion. On more than one occasion the pericardium has been tapped under the impression that the exudate was pleuritic. The flat tympany in the infrascapular region, the absence of well defined movable dulness, and the feeble, muffled sounds are indicative points. Followed from day to day there is rarely much difficulty, but it is different when a patient seen for the first time presents a large area of dulness in the antero-lateral region of the left chest, and there is no to and fro pericardial friction murmur. Many of the cases have been regarded as encapsulated pleural effusions.

A special difficulty exists in recognizing the large exudate in pneumonia. The effusion may be very much larger than the signs indicate, and the involvement of the adjacent lung and pleura is confusing. In at least three cases in our series we should have tapped the sac; post mortem the effusion was more than a litre.

The nature of the fluid can not positively be determined without aspiration; but a fairly accurate opinion can be formed from the nature of the primary disease and the general condition of the patient. In rheumatic cases the exudation is usually sero-fibrinous; in septic and tuberculous cases it is often purulent from the outset; in senile, nephritic, and tuberculous cases the exudate may be hæmorrhagic.

Treatment.—The patient should have absolute quiet, mentally and bodily,

Calvert says,

Superfine wheat off. - right to keep Cows - 5l. to 6l.		Double value
" Old. wheat " " " " - high - 4 - 5 lb.		

so as to reduce to a minimum the heart's action. Drugs given for this purpose, such as aconite or digitalis, are of doubtful utility. Local bloodletting by cupping or leeches is certainly advantageous in robust subjects, particularly in the cases of extension in pneumonia. The ice bag is of great value. It may be applied to the præcordia at first for an hour or more at a time, and then continuously. It reduces the frequency of the heart's action and seems to retard the progress of an effusion. Blisters are not indicated in the early stage.

When effusion is present, the following measures to promote absorption may be adopted: Blisters to the præcordia, a practice not so much in vogue now as formerly. It is surprising, however, in some instances, how quickly an effusion will subside on their application. Purges and iodide of potassium are of doubtful utility. The diet should be light, dry, and nutritious. The action of the kidneys may be promoted by the infusion of digitalis and potassium acetate.

With an effusion, so soon as signs of serious impairment of the heart occur, as indicated by dyspnœa, small, rapid pulse, dusky, anxious countenance, paracentesis, or incision of the pericardium, should be performed. With the sero-fibrinous exudate, such as commonly occurs after rheumatism, aspiration is sufficient; but when the exudate is purulent, the pericardium should be freely incised and freely drained. The puncture may be made in the fourth or fifth interspace, in or outside the nipple line. In large effusions the pericardium can be readily reached without danger by thrusting the needle upward and backward close to the costal margin in the left costo-xiphoid angle; or the needle may be introduced outside the left nipple line. The results of paracentesis of the pericardium have so far not been satisfactory. With an earlier operation in many instances and a more radical one in others —incision and free drainage, not aspiration, when the fluid is purulent— the percentage of recoveries will be greatly increased. Repeated tapping may be needed. One case of tuberculous effusion, tapped three times, recovered completely and was alive three years afterward.

CHRONIC ADHESIVE PERICARDITIS

(*Adherent Pericardium, Indurative Mediastinopericarditis*)

The remote prognosis in pericarditis is very variable. A large majority of these cases get well and have no further trouble, but in young persons serious results sometimes follow adhesions and thickening of the layers. As Sequira has pointed out, the danger is here directly in proportion to the amount of dilatation and weakening of the pericardium in consequence of the inflammation. The loss of the firm support afforded to the heart by the rigid fibrous bag in which it is inclosed is the important factor. There are two groups of cases of adherent pericardium.

(*a*) Simple adhesion of the peri- and epicardial layers, a common sequence of pericarditis, met with post mortem as an accidental lesion. It is not necessarily associated with disturbance in the function of the heart, which in a large proportion of the cases is neither dilated nor hypertrophied.

(*b*) Adherent pericardium with chronic mediastinitis and union of the

outer layer of the pericardium to the pleura and to the chest walls. This constitutes one of the most serious forms of cardiac disease, particularly in early life, and may lead to an extreme grade of hypertrophy and dilatation of the heart. The peritoneum may be involved with perihepatitis, cirrhosis, and ascites (Pick's disease).

Symptoms.—The symptoms of adherent pericardium are those of hypertrophy and dilatation of the heart, and later of cardiac insufficiency. G. D. Head in a careful study of 59 cases divides them into (1) a small silent group with no symptoms, (2) a larger group with all the features of cardiac disease, and (3) a group comprising 11 cases in his series in which the features were hepatic. To this last group much attention has been paid since Pick's description. The hepatic features dominate the picture and the diagnosis of cirrhosis of the liver is usually made. Recurring ascites is the special feature. One of my patients was tapped one hundred and twenty-one times. There is chronic peritonitis, with great thickening of the capsule of the liver and consequent contraction of the organ.

Diagnosis.—The following are important points in the diagnosis: *Inspection.*—A majority of the signs of value come under this heading. (*a*) The præcordia is prominent and there may be marked asymmetry, owing to the enormous enlargement of the heart. (*b*) The extent of the cardiac impulse is greatly increased, and may sometimes be seen from the third to the sixth interspaces, and in extreme cases from the right parasternal line to outside the left nipple. (*c*) The character of the cardiac impulse. It is undulatory, wavy, and in the apex region there is marked systolic retraction. (*d*) Diaphragm phenomena. Walter Broadbent has called attention to a very valuable sign in adherent pericardium. When the heart is adherent over a large area of the diaphragm there is with each pulsation a systolic tug, which may be communicated through the diaphragm to the points of its attachment on the wall, causing a visible retraction. This has long been recognized in the region of the seventh or eighth rib in the left parasternal line, but Broadbent called attention to the fact that it was frequently best seen on the left side behind, between the eleventh and twelfth ribs. This is a very valuable and quite common sign, and may sometimes be very localized. One difficulty is that, as A. W. Tallant has pointed out, it may occur in thin chested persons with great hypertrophy of the heart. Sir William Broadbent called attention to the fact that owing to the attachment of the heart to the central tendon of the diaphragm this part does not descend with inspiration, during which act there is not the visible movement in the epigastrium. (*e*) Diastolic collapse of the cervical veins, the so-called Friedreich's sign. This is not of much moment.

Palpation.—The apex beat is fixed, and turning the patient on the left side does not alter its position. On placing the hand over the heart there is felt a diastolic shock or rebound, which some have regarded as the most reliable of all signs of adherent pericardium.

Percussion.—The area of cardiac dulness is usually much increased. In a majority of instances there are adhesions between the pleura and the pericardium, and the limit of cardiac dulness above and to the left may be fixed and is uninfluenced by deep inspiration. This, too, is an uncertain sign, inasmuch as there may be close adhesions between the pleura and the peri-

cardium and between the pleura and the chest wall, which at the same time allow a very considerable degree of mobility to the edge of the lung.

Auscultation.—The phenomena are variable and uncertain. In the cases in children with a history of rheumatism endocarditis has usually been present. Even in the absence of chronic endocarditis, when the dilatation reaches a certain grade, there are murmurs of relative insufficiency, which, ~~as in one case I have recorded~~, may be present not only at the mitral but also at the tricuspid and pulmonary orifices. Theodore Fisher ~~has~~ called attention to the fact that there may be a well-marked presystolic murmur in connection with adherent pericardium. Occasionally the layers of the pericardium are united in places by strong fibrous bands, 5-7 mm. long by 3-5 mm. wide. In one such case Drasche heard a remarkable whirring, systolic murmur with a twanging quality.

The pulsus paradoxus, in which during inspiration the pulse-wave is small and feeble, is sometimes present, but it is not a diagnostic sign of either simple pericardial adhesion or of the cicatricial mediastino-pericarditis.

Cardiolysis, Brauer's operation, has been proposed for this condition and has been helpful in a few cases. Four or five centimetres of the fourth, fifth, and sixth left ribs with a couple of centimetres of the corresponding cartilages are resected, by which means the heart's action is less embarrassed. It is a justifiable procedure in selected cases—in, for example, a child with a very large, tumultuously acting heart, with much bulging of the chest.

II. OTHER AFFECTIONS OF THE PERICARDIUM

Hydropericardium.—The pericardial sac contains post mortem a few cubic centimetres of clear, citron colored fluid. In connection with general dropsy, due to kidney or heart disease, more commonly the former, the effusion may be excessive, adding to the embarrassment of the heart and the lungs, particularly when the pleural cavities are the seat of similar transudation. There are rare instances in which effusion into the pericardium occurs after scarlet fever with few, if any, other dropsical symptoms. Hydropericardium is frequently overlooked.

In rare cases the serum has a milky character—chylopericardium.

Hæmopericardium.—This condition is met with in aneurism of the first part of the aorta, of the cardiac wall, or of the coronary arteries, and in rupture and wounds of the heart. Death usually follows before there is time for the production of symptoms other than those of rapid heart failure due to compression. In rupture of the heart the patient may live for many hours or even days with symptoms of progressive heart failure, dyspnœa, and the physical signs of effusion.

In the pericarditis of tuberculosis, of cancer, of ~~Bright's disease~~ n.p.h., and of old people the inflammatory exudate is often blood stained.

Pneumopericardium.—This is an excessively rare condition, of which Walter James was able to collect only 38 cases in 1903. ~~I~~ have met with but one instance, from rupture of a cancer of the stomach. Perforation of the sac occurred in all but 5, in which the gas bacillus was the possible cause, as in Nicholl's case, ~~at the Royal Victoria Hospital, Montreal,~~ this organism

was isolated. Seven cases were due to perforation of the œsophagus and eight to penetrating wounds from without. The physical signs are most characteristic. A tympany replaces the normal pericardial flatness. On auscultation there is a splashing, gurgling, churning sound, called by the French *bruit de moulin*. This was described in 19 of the cases collected by James. Of the 38 cases, 26 died.

Calcified Pericardium.—This remarkable condition may follow pericarditis, particularly the suppurative and tuberculous forms; occasionally it extends from the calcified valves. It may be partial or complete. Of 59 cases collected by A. E. Jones, in 38 there were no cardiac symptoms. Adherent pericardium was diagnosed in one case. Jones's careful study shows that the condition is usually latent and unrecognized.

B. DISEASES OF THE HEART

I. FUNCTIONAL AFFECTIONS OF THE HEART

1. PALPITATION

In health we are unconscious of the action of the heart. One of the first indications of debility or overwork is the consciousness of the cardiac pulsations, which may, however, be perfectly regular and orderly. This is not palpitation. The term is properly limited to irregular or forcible action of the heart perceptible to the individual. The condition of extra-systole described in the next section is present in many cases.

Etiology.—The expression "perceptible to the individual" covers the essential element in palpitation of the heart. The most extreme disturbance of rhythm, a condition even of what is termed *delirium cordis*, may be unattended with subjective sensations of distress, and there may be no consciousness of disturbed action. On the other hand, there are cases in which complaint is made of the most distressing palpitation and sensations of throbbing, in which the physical examination reveals a regularly acting heart, the sensations being entirely subjective. We meet with this symptom in a large group of cases in which there is increased excitability of the nervous system. Palpitation may be a marked feature at the time of puberty, at the climacteric, and occasionally during menstruation. It is a very common symptom in hysteria and neurasthenia, particularly in the form of the latter which is associated with dyspepsia. Emotions, such as fright, are common causes of palpitation. It may occur as a sequence of the acute fevers. Females are more liable to the affection than males.

In a second group the palpitation results from the action upon the heart of certain substances, such as tobacco, coffee, tea, and alcohol. And, lastly, palpitation may be associated with organic disease of the heart, either of the myocardium or of the valves. As a rule, however, it is a purely nervous phenomenon—seldom associated with organic disease—in which the most violent action and the most extreme irregularity may exist without that subjective element of consciousness of the disturbance which constitutes the essential feature of palpitation.

Phrenocardia. pain in card. reg. palpitation + resp. d ? adhes.
to left of thor.

pain to left of card. dullness. sometimes bdw apex. corresponds to attach. of diaph. & double apex-radic cardi. of diaphragm.
Ant hungry. tricuspid & the deep recess. result from lowering of diaph.
on left side. ∴ heart also sinks. = deep heart murmur
∴ = cardiac murmur palpitation most common complaint

Gallop. rhythm. may be due to excessive pumping of ventricle. accords ad.
press much blood into vent. - distending it abruptly. This causes a
sound. A presystolic bruit pulse often accompanies it in nephritis with
gallop rhythm.

Functional murmurs. Diagnosis.
Mus. when organic are unusual - soft. not carried.
Vary day to day with exercise + rest - not reg. with each systole.
Ms. most marked valvular - mid. by degree, endocardial.
affect endocard. or ☆ mur. may appear.
Pressure on eyeballs arrests functional murmurs. Then
have bet. true + func. murmurs - again arrested by ac. murmurs
pulse full.
Org. murmurs not arrested by pr. on eyeballs.

Tubi. capable heart - beat 70 valvular to 80 standing also aff. cardiac.
H.R. little change standing.

Diag. of nervous Card. Dist.

Lymphocytosis - imp. in diag. of toxic affections from thyroid.

helpful in diag. bet. organic & functional cardiac.

Epinephrin - 2 drops of 1-1000 soln. in eye. (watch for ↑ normal)

causes mydriasis - prompt. marked - test for cerebral trauma.

if thyroid abnormal.

The irritable heart described by Da Costa, which was so common among the young soldiers during the civil war, is a neurosis of this kind. The chief symptoms were palpitation with great frequency of the pulse on exertion, a variable amount of cardiac pain, and dyspnœa. The factors at work in producing this condition appeared to be the mental excitement, the unwonted muscular exertion associated with the drill, and diarrhœa. The condition is not infrequent in civil life among young men, and when persistent it may lead to hypertrophy of the heart.

Symptoms.—In the mildest form, such as occurs during a dyspeptic attack, there are slight fluttering of the heart and a sense of what patients sometimes call "goneness." In more severe attacks the heart beats violently, its pulsations against the chest wall are visible, the rapidity of the action is much increased, the arteries throb forcibly, and there is a sense of great distress. In some instances the heart's action is not at all quickened. The most striking cases are in neurasthenic women, in whom the mere entrance of a person into the room may cause the most violent action of the heart and throbbing of the peripheral arteries. The pulse may be rapidly increased until it reaches 150 or 160. A diffuse flushing of the skin may appear at the same time. After such attacks there may be the passage of a large quantity of pale urine. In many cases of palpitation, particularly in young men, the condition is at once relieved by exertion. A patient with extreme irregularity of the heart may, after walking quickly 100 yards or running upstairs, return with the pulse perfectly regular. This is not infrequently seen, too, in the irregular action of the heart in mitral valve disease.

The physical examination of the heart is usually negative. The sounds, the shock of which may be very palpable, are on auscultation clear, ringing, and metallic, but not associated with murmurs. The second sound at the base may be greatly accentuated. A murmur may sometimes be heard over the pulmonary artery or even at the apex in cases of rapid action in neurasthenia or in severe anæmia. The attacks may be transient, lasting only for a few minutes, or may persist for an hour or more. In some instances any attempt at exertion renews the attack. Sometimes in vigorous young adults who are upset nervously, especially after exertion or during excitement, the signs of mitral stenosis are simulated. There is a systolic shock preceded by a suggestion of a thrill. On auscultation it may be difficult to decide whether or not there is a short presystolic murmur. A short period of observation usually removes the uncertainty.

The *prognosis* is usually good, though it may be extremely difficult to remove the conditions underlying the palpitation.

2. ARRHYTHMIA

The work of Gaskell and of Engelmann on the functions of the heart muscle, and the clinical studies of James Mackenzie, Wenckebach, and others, have modified the older views of the neurogenic cardiac mechanism with its musculo-motor nerve centres upon which the higher centres played through the vagi and the sympathetic nerves. The source of the action of the heart is now placed in the muscle itself—myogenic—and Gaskell describes as its functions rhythmicity, excitability, contractility, conductivity, and tonicity; "that

is to say, the muscular fibres of the heart possess the power of rhythmically creating a stimulus, of being able to receive a stimulus, of responding to a stimulus by contracting, of conveying the stimulus from muscle fibre to muscle fibre, and of maintaining a certain ill-defined condition called tone." Wenckebach and James Mackenzie have studied the disturbances of these functions of the heart clinically, and have endeavored to classify them in harmony with the myogenic theory. I am indebted to Erlanger for the following classification based on that of Wenckebach:

I. *Arrhythmia resulting from decreased conductivity in the auriculo-ventricular junction—heart-block.* Characteristics: Auricular rhythm perfect, rate normal or accelerated; ventricular rhythm may or may not be perfect; if perfect its rate will be one half of that of the auricles, or less; if not perfect the irregularities will bear some direct relation to the contractions of the auricles.

A. Partial heart-block: (1) Occasional ventricular silence; (2) regularly recurring ventricular silence, either one ventricular beat missed in 7, 6, 5, 4, etc., auricular beats, or a 2:1, 3:1, 4:1 rhythm, or either of these alternating.

B. Complete heart-block: Auricular and ventricular rhythms perfect but independent.

C. Paroxysmal bradycardia (Stokes-Adams disease) affecting the ventricular rate alone.

II. *Arrhythmia resulting from increased irritability of the heart.*

A. Ventricular extra-systoles, characterized by an early systole, which is associated with the phenomena of a retrograde impulse. There may be one or more extra-systoles following a normal systole; when regularly recurring, one or more extra-systoles after 5, 4, 3, 2, or 1 normal systoles, the last giving the bigeminal or trigeminal pulse, or there may be irregularly recurring extra-systoles causing delirium cordis.

B. Auricular extra-systoles.

C. Nodal extra-systoles, causing the auricle and ventricle to contract at nearly the same time.

III. *Arrhythmia resulting from a deficiency of contractility in the cardiac muscle.*

A. Alternating pulse.

B. Omissions of ventricular systole, e. g., in the halved rate seen after large doses of digitalis.

IV. *Arrhythmia resulting from the influence of extrinsic nerves upon the heart-rate.* (1) Vagus effect, and (2) accelerator effects. These are seen in the irregularities synchronous with respiration in the youthful type of irregularity, in "sinus" irregularity, and in certain forms of paroxysmal tachycardia (vagal). Extra-systoles seen in nervous persons are probably due to a slight excess of accelerator action.

V. *Arrhythmia resulting from disturbed diastolic filling of the heart.*

A. Disturbed filling resulting from violent respiratory movements: may give the paradoxical pulse.

B. Disturbed filling from adherent pericardium or mediastinal tumor: may give the paradoxical pulse.

Intermittency: Extra-systoles.—The commonest type of arrhythmia is that now known as the extra-systole, to explain which it must be remembered that to a stimulus strong enough to set up a contraction the heart answers with all the contractility of which it is capable at the moment (Bowditch's law of maximal contraction). A second property of the heart muscle is that it possesses a "refractory phase" in which normally it is not excitable, or answers only to very strong stimuli. During this refractory stage, beginning shortly before the systole and continuing a short time after it, the heart is inexcitable. When not refractory it may again contract during this phase and produce an

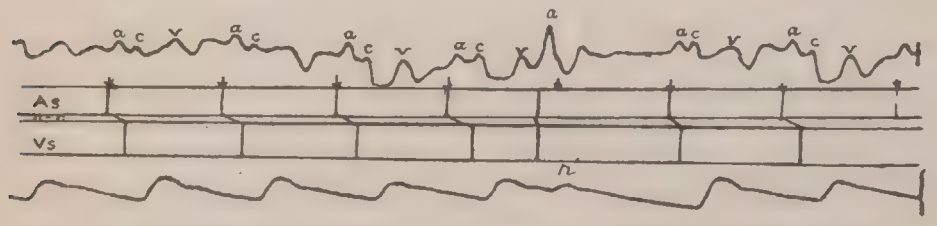

FIG. 1.—A "NODAL" EXTRA-SYSTOLE. The auricular and ventricular systoles are premature and simultaneous (Mackenzie).

extra-systole, which is followed by a long pause. Engelmann explains this long pause as follows: "In consequence of the extra-systole the ventricle is still in the refractory stage when the next physiological stimulus reaches it. This stimulus, therefore, has no effect, no contraction takes place, and it is not till the next stimulus after it that a contraction can again be produced. Thus the normal systole that would follow the extra-systole is missed; then the first systole that comes after the compensatory pause occurs exactly at the moment at which it would have occurred had no extra-systole preceded it" (Wenckebach). The irregularity, inequality, and intermission of the pulse as

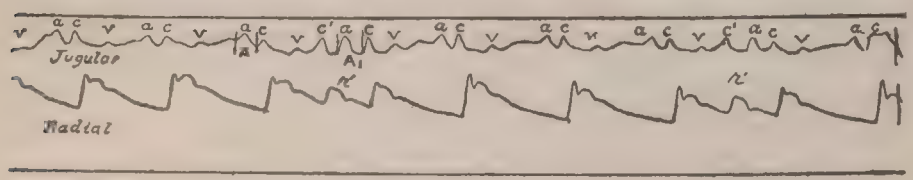

FIG. 2.—EXTRA-SYSTOLES OF VENTRICULAR TYPE AT *c'* and *r'* (Mackenzie).

met with in every day clinical experience are largely due to the occurrence of these extra-systoles, which may present all sorts of combinations and groupings, bigeminal, trigeminal, etc., depending upon whether the extra pulse beats are perceptible or not. And yet in spite of this most extreme irregularity there may be no actual pathological change, and so far as the maintenance of the circulation is concerned the heart may be acting in a most satisfactory manner. Patients may feel the extra-systole as a definite thud, and the compensatory pause is perceptible, but very often there are no subjective sensations.

Extra-systoles occur at all ages and under the most varied conditions in health and disease. Mackenzie recognizes a youthful and an adult type of arrhythmia, which in the latter is due chiefly to the presence of extra-systoles. There are several classes of cases. The arrhythmia may be a life-long condition. Without any recognizable disease, without any impairment of the action of the heart, there is permanent irregularity. This may be a peculiarity of the heart-muscle of the individual, who has extra-systole for the same reason—physiological but not well understood—as the dog and horse, in which animals this phenomenon is common. The late Chancellor Ferrier, of McGill University, who died at the age of eighty seven, had an extremely irregular heart action for the last fifty years of his life. I know several men who have

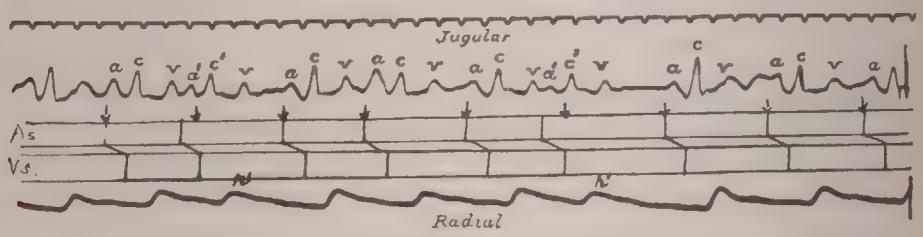

FIG. 3.—EXTRA-SYSTOLES (a') FOLLOWED BY VENTRICULAR CONTRACTIONS (c', r'). The arrows in the diagram represent the sinus stimulation, and the long pauses after the extra-systoles are seen to be due to the fact that the auricle did not respond to the sinus stimulation after the extra-systole (as, auricular systole vs. ventricular systole) (Mackenzie).

had for many years irregularity without the slightest discomfort. In debilitated and neurasthenic persons there may be an irritable weakness of the heart associated with extra-systole, and palpitation of a most distressing character. In a second group toxic agents, as tobacco, tea, coffee, or the poisons of the infectious diseases or those originating in the intestines or metabolic poisons cause arrhythmia. Even reflexly, as in flatulent dyspepsia, extra-systoles may arise. Thirdly, a high blood pressure can set up extra-systoles; also change in posture. And, lastly, organic disease of the heart itself, "dilatation, inflammation, poor blood supply to the muscle, overexertion, can all supply stimuli to set up extra-systoles either directly or reflexly" (Wenckebach). Too much stress should not be laid upon arrhythmia *per se* in the absence of organic disease.

3. FIBRILLATION OF THE HEART

(*Cardiac Flutter, Nodal Rhythm, Pulsus Irregularis Perpetuus*)

This common manifestation of cardiac irregularity is exceedingly important to recognize clinically. In the most pronounced form it is seen in the last stages of mitral stenosis, in which the pulse shows extreme irregularity, which, when once established, seldom returns to normal. A study of its features in this condition gave Mackenzie the clue to its explanation. He found that in certain cases the transition from regular to irregular pulse of this type occurred with suddenness, and that, whereas before the irregularity supervened the jugular pulse showed the normal features in the presence of auricular carotid and ventricular waves, with a marked presystolic murmur and thrill at the apex, after the irregularity was established, the auricular

Vagus may be capable of causing extra systoles. Extra sys. may be caused by pressure on the eyeball (Oculo-cardiac reflex)
Ex. sys. are most common in neurosis (mental reflex). In some cases mere skin-pain was enough to cause. If vagus concerned then atropin may be indicated. May toxins or vagus be cause of Ex sys. at times?

Digitalis [illegible]

Oculo-cardiac reflex - with pulsation present in eyeball the heart rhythm changed to that typical of pronounced mitral stenosis
Volt modification of rhythm + unbinding of vagus influence may help in diag. of organic disease
Pressure on eyeballs - between [illegible]

wave disappeared from the jugular pulse and the presystolic murmur from the apex. The inference drawn from these facts was that the right auricle of the heart was so dilated as to prevent the formation of a normal auricular contraction. The stimulus normally produced at or near the superior vena cava was prevented from passing down the junctional tissues to the ventricle; hence the stimulus arose in another and slightly less excitable part of the heart, which Mackenzie supposed to be the auriculo-ventricular node of Tawara on the right side of the auricular septum. Complete proof of the cause of this condition has been supplied by Lewis, who found that patients with this irregularity showed in galvanometric tracings from the auricle numerous small and continuous waves, exactly similar to those obtained in the dog after fibrillation of the auricle has been induced by faradic stimulation of the appendix, of the right auricle, or by ligation of the right coronary artery. Very small auricular beats are occasionally to be seen in the jugular tracings from such patients. As no co-ordinated contraction of the auricle is present, the arrival of the stimuli at the junctional tissues has no orderly sequence, the ventricle is stimulated without regularity, and in consequence the pulse is irregular.

True nodal rhythm is a comparatively rare condition, in which the stimulus production arises in the junctional tissues; the diagnostic feature is the simultaneous contraction of auricle and ventricle as shown by the superposition of the *a* and *c* waves of the jugular pulse. The radial pulse in this condition is regular.

Auricular fibrillation forms a large proportion of the cases showing cardiac irregularity. Of 114 cases of all forms of irregularity studied by Lewis 57 were of this type. Of etiological factors the most important are mitral stenosis, whether in the rheumatic form or that seen in women with no history of rheumatism and cardio-stenosis. The average age of onset in those with a previous history of rheumatism is 30 to 40; in the non-rheumatic group it is between 50 and 60. In the older patients it may show a paroxysmal form or at least prolonged intermissions.

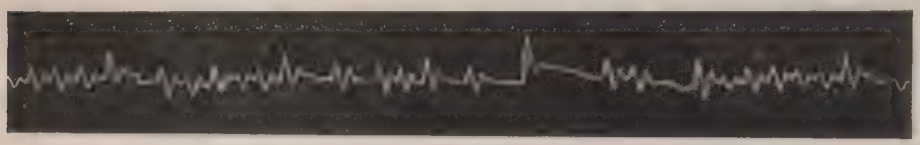

FIG. 4.—PULSE TRACING FROM A CASE OF AURICULAR FIBRILLATION.

The symptoms due to the fibrillation itself cannot be appreciated in the cases of mitral stenosis, but may be seen in those not so accompanied. Here the symptoms may be absent, or consist in some limitation in the field of cardiac response: the patient more easily becomes breathless on exertion, and there may be a tendency to much greater fatigue. The inception of fibrillation may be associated with great dyspnœa, orthopnœa, and the features of right heart failure. The condition may last for many years when once established, and the patient may be able to follow an arduous occupation. An unduly grave view must not, therefore, be taken, unless there be marked evidence of cardiac failure. The patient should be cautioned against undue exertion.

4. RAPID HEART—TACHYCARDIA

The rapid action may be perfectly natural. There are individuals whose normal heart action is at 100 or even more per minute. Emotional causes, violent exercise, and fevers all produce great increase in the rapidity of the heart's action. The extremely rapid action which follows fright may persist for days or even weeks. Traube reports an instance in which, after violent exercise, the rapidity of the heart continued. Cases are not uncommon at the menopause.

There are cases again in which the condition can hardly be termed a neurosis, since it depends upon definite changes in the pneumogastrics or in the medulla. Cases have been reported in which tumor or clot in or about the medulla or pressure upon the vagi has been associated with heart hurry. Some of the cases of frequent action of the heart in women have been thought to be due to reflex irritation from ovarian or uterine disease. Other cases are almost certainly due to lesions of the heart itself and are now and then seen subsequent to an influenzal attack; young and old persons are affected. The tachycardia may persist for months or indefinitely, and there is serious interference with the amount of muscular exertion such persons can take; in addition there is a sense of weakness and sometimes fainting attacks.

Paroxysmal Tachycardia.—Modern methods enable us to subdivide the cases of paroxysmal tachycardia into three groups, corresponding to the types of extra-systole—the auricular, the nodal, and the ventricular. In the auricular type there is a well marked auricular wave in the venous pulse in its normal relation to the carotid wave; in the nodal type the auricular and carotid wave occur at the same time, giving a large double wave in the venous tracing; the third type has not been seen in man, but from experimental work on the dog (Lewis) it may be predicted that it will be found. These three types are due to an irritable focus in the cardiac musculature, the auricular in the auricular muscle, the nodal in the functional (auriculo-ventricular) tissues which form the muscular bridge between the auricle and the ventricle, and the ventricular in the ventricular muscle. In many cases it may be due to slight or transitory ischæmia from a sclerosed artery or one whose muscle is liable to spasm, for Lewis has shown that on tying the right coronary artery in the dog attacks of ventricular tachycardia are almost invariably seen. "A hyper-excitability of some focus in the cardiac musculature as the direct exciting cause of paroxysmal tachycardia is a probable explanation of its production. The degree of response to stimulation depends not only on the intensity of the stimulus, but also on the excitability of the tissue itself. In the known increased excitability of the auricle as compared to the ventricle may possibly be found an explanation of the increased frequency of auricular over other forms of tachycardia" (Lea).

The pulse rate may reach 200 and over. The cases are not common. The attack may be quite short and persist only for an hour or so. A patient at the Philadelphia Infirmary for Nervous Diseases was attacked every week or two; the pulse would rise to 220 or 230, and there were such feelings of distress and uneasiness that the patient always had to lie down. There may be, however, no subjective disturbance, and in another case the patient was able

Auricular flutter in rheumatic carditis

Suggestions. 1. Great dyspnoea. sudden & well-mk change in card. condition
2. Triple or galloping cardiac rhythm.
3. Pericarditis without effusion & with great resp. distress
vs. more marked about pain. than vent.
4. Dyspnoea, cyanosis & enlarge. of liver subj. prop. to degree of heart
5. Pulsus alternans.

Oculo- cardiac reflex in fr.

fr. in eyeball 15 sec - refractory

swallowing "channels" of brain

oculo-cardiac reflex - pressure on eyeball slows pulse.

afferent impulses from eye by ophthalmic branch of V to the gasserian ganglion thy larger root to nucleus of cells a few with the middle the superior peduncles cerebellum, just inferior vagi in lower part of floor of 4 ventricle - centres in medulla cells respiratory of heart

- an acceleration or retardation of 10 beats abnormal.

- Note about is taken (always with eye not pupil)

about is right

to walk about during the paroxysm and had no dyspnœa. One of the most remarkable cases is reported by H. C. Wood. A physician in his eighty-seventh year had had attacks at intervals from his thirty-seventh year. The onset was abrupt and the pulse would rapidly rise to 200 a minute. For more than twenty years the taking of ice-water or strong coffee would arrest the attacks. Bouveret, who has analyzed a number of cases of this essential or idiopathic form, finds that a permanent cure is rare, and that the patients suffer for ten or more years. Four instances terminated fatally from heart-failure. One of the most remarkable features is the abruptness of onset and the abruptness with which an attack may end. One of my patients had recurring attacks lasting ten to thirty days, and the heart would suddenly "flop," as she expressed it, the rate falling from 180 to 80 or 90 per minute.

5. SLOW HEART—BRADYCARDIA

Slow action of the heart is sometimes normal and may be a family peculiarity. Napoleon is stated to have had a pulse of only 40 per minute.

In any case of slow pulse it is important first to make sure that the number of heart and arterial beats correspond. In many instances this is not the case, and with a radial pulse at 40 the cardiac pulsations may be 80, half the beats not reaching the wrist. The heart contractions, not the pulse wave, should be taken into account.

Physiological Bradycardia.—As age advances the pulse rate becomes slow. In the puerperal state the pulse may beat from 44 to 60 per minute, or may even be as low as 34. It is seen in premature labor as well as at term. The explanation of its occurrence at this period is not clear. Slowness of the pulse is associated with hunger. Bradycardia depending on individual peculiarity is extremely rare.

Pathological bradycardia, which is met with under the following conditions: (*a*) In convalescence from acute fevers. This is extremely common, particularly after pneumonia, typhoid fever, rheumatic fever, and diphtheria. It is most frequently seen in young persons and in cases which have run a normal course. (*b*) In diseases of the digestive system, such as chronic dyspepsia, ulcer or cancer of the stomach, and jaundice. (*c*) In diseases of the respiratory system. Here it is by no means so common, but it is seen not infrequently in emphysema. (*d*) In diseases of the circulatory system. Excluding all cases of irregularity of the heart, bradycardia is not common in diseases of the valves. It is most frequently seen in fatty and fibroid changes in the heart, but is not constant in them. (*e*) In diseases of the urinary organs. It occurs occasionally in nephritis and may be a feature of uræmia. (*f*) From the action of toxic agents. It occurs in uræmia, poisoning by lead, alcohol, and follows the use of tobacco, coffee, and digitalis. (*g*) In constitutional disorders, such as anæmia, chlorosis, and diabetes. (*h*) In diseases of the nervous system. Apoplexy, epilepsy, the cerebral tumors, affections of the medulla, and diseases and injuries of the cervical cord may be associated with very slow pulse. In general paresis, mania, and melancholia it is not infrequent. (*i*) It occurs occasionally in affections of the skin and sexual organs, and in sunstroke, or in prolonged exhaustion from any cause.

6. HEART-BLOCK. STOKES-ADAMS DISEASE

The impulse causing the heart to beat originates at the venous end of the heart and is transmitted in such a way that the auricles contract first, the ventricles a moment later, the impulse being propagated like a peristaltic wave through the heart walls. In passing from the auricle to the ventricle the stimulus traverses a narrow band of muscle, the only demonstrable muscular connection between the venous and arterial chambers. In the adult heart this auriculo-ventricular bundle of His is 18 mm. long, 2.5 mm. broad, and 1.5 mm. thick; it arises in the septum of the auricles below the foramen ovale and passes downward and forward through the trigonum fibrosum of the auriculo-ventricular junction, where it comes into close relation with the mesial leaflet of the tricuspid valve. Passing along the upper edge of the

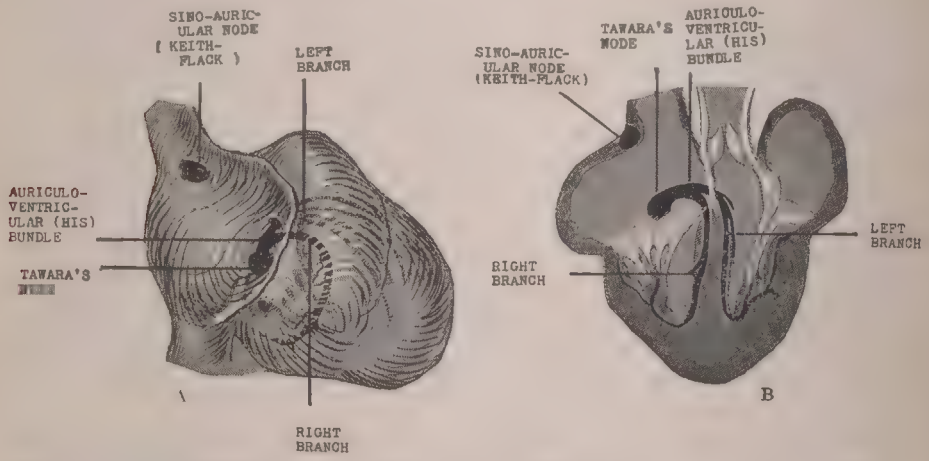

FIG. 5.—DIAGRAM SHOWING THE SINO-AURICULAR NODE AND THE AURICULAR BUNDLE. A, viewed from the right; B, cross section of the heart, viewed from the front. (Kindness of A. D. Hirschfelder.)

muscular septum, just where it joins with the posterior edge of the membranous septum, it radiates from this point throughout the heart as the junctional system of Tawara. In the dog destruction of the bundle prevents the passage from the auricle to the ventricle of the impulse which normally causes the ventricles to contract. They immediately assume a rate of beating which is very much slower than that of the auricles and is totally independent, as they possess their own automatic rhythmicity. Under ordinary circumstances this inherent rhythmicity can not manifest itself because the much more rapidly beating venous end of the heart sets the pace for the sluggish arterial end. But if the auricular impulse is blocked, the ventricles, released from the control of their normal pace maker, assume their own rate. This condition has been called complete heart-block. By an ingenious contrivance Erlanger has been able in the dog gradually to compress the auriculo-ventricular bundle and produce various stages of this condition, namely, one ventricular silence with every other auricular beat, giving a 2:1 rhythm, and proceeding to a 3:1 and a 4:1 rhythm. Finally, complete block may result,

in which no impulses pass from the auricles, but the ventricles beat with their own inherent rate, which Erlanger estimates from a study of cases of heart-block in my wards, to be about 23 to 28 beats to the minute in man. The explanation of the phenomenon is based upon one suggested by Gaskell. The bundle of His, like all muscle tissue, becomes fatigued when it is made to contract repeatedly. Under normal circumstances sufficient time elapses between successive beats to permit the bundle to return to its normal state, but when from injury or any cause the irritability of the bundle is greatly reduced, it may not react to the auricular stimulus, which thus fails to reach the ventricles. Occasionally while compressing the auriculo-ventricular bundle in the dog, the ventricle alone may suddenly stop beating for as long as twenty seconds. The explanation is here to be sought for the syncopal attacks in Stokes-Adams disease. In this condition the relaxed ventricles are distended rhythmically by the beats of the auricles until the distention may be extreme. The veins become engorged and pulsate synchronously with the auricles. Each of the infrequent contractions of the ventricles relieves the condition temporarily. When the heart-block is complete the vagi still exert their normal control over the rate of the auricles, but they have lost almost completely their influence over the ventricles, and in Stokes-Adams disease we find the pulse rate is little influenced by conditions which normally alter it, as exercise, posture, etc.

Clinically Stokes-Adams disease presents three features: (*a*) slow pulse, usually permanent, but sometimes paroxysmal, falling to 40, 20, or even 6 per minute; (*b*) cerebral attacks—vertigo of a transient character, syncope, pseudo-apoplectiform attacks or epileptiform seizures; (*c*) visible auricular impulses in the veins of the neck, as noted by Stokes—the beats varying greatly; a 2:1 or 3:1 rhythm is the most common. There are several groups of cases. It is usually a senile manifestation associated with arterio-sclerosis. The cases in young adults and middle aged men are often myocardial and of syphilitic origin. There is a neurotic group in which all the features may be present, and in which post mortem no lesions have been found (Edes and Councilman). In the attacks of slow pulse in this group the auricular as well as the ventricular rate may be slow and equal, the normal sequence of events being preserved; the origin of the condition is probably vagal. The outlook in this class of cases is good; in the others it is a serious disease and usually fatal, though it may last for many years. The cerebral attacks are due to anæmia of the brain or of the medulla in consequence of the imperfect ventricular action. In one of ~~my~~ cases Baetjer could see with the fluoroscope the more frequent contraction of the auricles.

TREATMENT OF PALPITATION AND ARRHYTHMIA

An important element in many cases is to get the patient's mind quieted, and he can be assured that there is no actual danger. The mental element is often very strong. In palpitation, before using medicines, it is well to try the effect of hygienic measures. As a rule, moderate exercise may be taken with advantage. Regular hours should be kept, and at least ten hours out of the twenty four should be spent in the recumbent posture. A tepid bath may be taken in the morning, or, if the patient is weakly and nervous, in the

evening, followed by a thorough rubbing. Hot baths and the Turkish bath should be avoided. The dietetic management is most important. It is best to prohibit alcohol, tea, and coffee absolutely. The diet should be light and the patient should avoid taking large meals. Articles of food known to cause flatulency should not be used. If a smoker, the patient should give up tobacco. Sexual excitement is particularly pernicious, and the patient should be warned specially on this point. For the distressing attacks of palpitation which occur with neurasthenia, particularly in women, a rigid Weir Mitchell course is the most satisfactory. It is in these cases that we find the most distressing throbbing in the abdomen, which is apt to come on after meals, and is very much aggravated by flatulency. The cases of palpitation due to excesses or to errors in diet and dyspepsia are readily remedied by hygienic measures.

A course of iron is often useful. Strychnia is particularly valuable, and is perhaps best administered as the tincture of nux vomica in large doses. Very little good is obtained from the smaller quantities. It should be given freely, 20 minims (1.3 c. c.) three times a day.

If there is great rapidity of action, aconite may be tried. There are cases associated with sleeplessness and restlessness which are greatly benefited by bromide of potassium. Digitalis is very rarely indicated, but in obstinate cases it may be tried with the nux vomica. Ammonium bromide is very efficacious in the tachycardias and arrythmias of nervous people.

Cases of heart hurry are often extremely obstinate, as may be judged from the case of the physician reported by H. C. Wood, in whom the condition persisted in spite of all measures for fifty years. The bromides are sometimes useful; the general condition of neurasthenia should be treated, and during the paroxysm an ice bag may be placed upon the heart, ~~or Leiter's coil, through which ice water is passed.~~ Electricity, in the form of galvanism, is sometimes serviceable, and for its mental effect the Franklinic current. For the condition of slow pulse but little can be done. A great majority of the cases are not dangerous.

II. AFFECTIONS OF THE MYOCARDIUM

1. HYPERTROPHY

Varieties.—The heart enlarges to meet a demand for extra work, either general, as in the continuous strain of athletics (the hypertrophy of work), or special to combat a deficiency of cardiac structure, such as a damaged valve. There are two forms, one in which the cavity or cavities are of normal size, and the other in which the cavities are enlarged and the walls increased in thickness (eccentric hypertrophy). The so-called concentric hypertrophy in which there is diminution of the size of the cavity with thickening of the walls is, as a rule, a post mortem change.

The enlargement may affect the entire organ, or one side, or only one chamber. Naturally, as the left ventricle does the chief work the change is most frequently found here. Though its production is assisted by adequate nutrition, hypertrophy may appear even under conditions of starvation, given otherwise healthy organs. In the debilitated the limits to which hypertrophy

may progress are small, though very marked hypertrophy is sometimes seen in the aged.

HYPERTROPHY OF THE LEFT VENTRICLE ALONE, or with general enlargement of the heart, is brought about by—

Conditions affecting the heart itself: (*a*) Disease of the aortic valve; (*b*) mitral insufficiency; (*c*) pericardial adhesions; (*d*) sclerotic myocarditis; (*e*) disturbed innervation with overaction, as in exophthalmic goitre, in long-continued nervous palpitation, and as a result of the action of certain articles, such as tea, coffee, tobacco, and probably alcohol, as in the Munich beer heart. In all of these the work of the heart is increased. In the case of the valve lesions the increase is due to the increased intraventricular pressure; in the case of the adherent pericardium and myocarditis, to direct interference with the symmetrical and orderly contraction of the chambers.

Conditions acting upon the blood-vessels: (*a*) General arterio-sclerosis, with or without renal disease, especially sclerosis of the aorta, the renal arteries, and the vessels of the splanchnic area; (*b*) all states of increased arterial tension induced by the contraction of the smaller arteries under the influence of certain toxic substances, which, as Bright suggested, "by affecting the minute capillary circulation, render great action necessary to send the blood through the distant subdivisions of the vascular system"; (*c*) prolonged muscular exertion, which enormously increases the blood pressure in the arteries; (*d*) narrowing of the aorta, as in the congenital stenosis.

HYPERTROPHY OF THE RIGHT VENTRICLE is met with under the following conditions—

(*a*) *Lesions of the mitral valve,* either incompetence or stenosis, which act by increasing the resistance in the pulmonary vessels. (*b*) *Pulmonary lesions,* obliteration of any number of blood vessels within the lungs, such as occurs in emphysema or cirrhosis, is followed by hypertrophy of the right ventricle. (*c*) *Valvular lesions* on the right side occasionally cause hypertrophy in the adult, not infrequently in the fetus. (*d*) *Chronic valvular disease of the left heart and pericardial adhesions* are sooner or later associated with hypertrophy of the right ventricle.

In the auricles simple hypertrophy is never seen; there is always dilatation with hypertrophy. In the left auricle the condition develops in lesions at the mitral orifice, particularly stenosis. The right auricle hypertrophies when there is greatly increased blood pressure in the lesser circulation, whether due to mitral stenosis or pulmonary lesions. Narrowing of the tricuspid orifice is a less frequent cause.

Symptoms.—There may be no complaint attributable to the hypertrophy, and if associated with renal disease or arterio-sclerosis there may be a marked sense of well-being. If, however, the cardiac defect be not fully compensated, the patient may complain of slight giddiness, headache, a sense of palpitation in the thorax, and some dyspnœa on exertion.

In hypertrophy of the right auricle the venous pulsation in the neck may be more evident, and a tracing may show a marked increase in the size of the auricular wave. An increase in dulness to the right of the sternum in the third and fourth interspaces may be detected, and on very rare occasions a sound preceding that of the ventricle over that area. Hypertrophy of the right ventricle causes a slight bulging of the costal angle with a positive

instead of a negative pulsation at this spot. The apex beat may be diffuse, as the enlarged right ventricle prevents the left ventricle from coming into contact with the chest wall. The venous pulsation in the neck is usually marked, and the first sound over the tricuspid area louder than normal. Hypertrophy of the left auricle, which is seldom marked and never unassociated with dilatation, may be detected occasionally by dulness toward the base of the left lung behind; it is easily diagnosed by the extension backward of the cardiac shadow in oblique illumination of the chest by the X-rays. Hypertrophy of the left ventricle is usually easy to diagnose. There is a forcible impulse at the apex beat, both visible and palpable. This impulse may cause a movement of a large area of the chest wall. The apex beat, if there be only slight dilatation, is usually displaced downward, and is found in the 6th and 7th spaces; but if the dilatation be marked, the apex beat becomes more diffuse and is found well outside the nipple line in the 4th, 5th, and 6th spaces. The first sound is usually marked and has been described by Michell as "L-lumb"; sometimes it has a distinct booming sound. The second sound at the base is accentuated. The pulse is full and of high tension at the height of the ventricular impulse. The blood pressure is raised.

2. DILATATION

As with other hollow muscular organs, the size of the chambers of the heart varies greatly within normal limits. Dilatation may be an acute process and quite transitory, as after severe muscular effort, or it may be chronic, in which case it is associated with hypertrophy. Not always, however; there is an extraordinary heart in the McGill College Museum showing a parchment like thinning of the walls with uniform dilatation of all the chambers; in places in the right auricle and ventricle only the epicardium remains. Dilatation is pathological only when permanent. Increase in capacity means increased work for the walls and in consequence hypertrophy to meet the demand.

Etiology.—Two important causes combine to produce dilatation—increased pressure within the cavities and impaired resistance, due to weakening of the muscular wall—which may act singly, but are often combined. A normal wall may yield under a heightened blood pressure, or a weakened wall may yield to a normal distending force, the weakened wall being due either to structural change in the cardiac muscle, or to a diminution of its natural tonus.

(*a*) HEIGHTENED ENDOCARDIAC PRESSURE results either from an increased quantity of blood to be moved, or an obstacle to be overcome, and is the more frequent cause of weakening. It does not necessarily bring about dilatation; simple hypertrophy may follow, as in the early period of aortic stenosis, and in the hypertrophy of the left ventricle in ~~Bright's disease~~ nephritis.

The size of the cardiac chambers varies in health. With slow action of the heart the dilatation is complete and fuller than it is with rapid action. Moderate exertion in a normal heart, or even prolonged exertion in a well-trained heart, lessens the heart size, but in conditions of ill health dilatation occurs. Physiologically, the limits of dilatation are reached when the chamber does not empty itself during the systole. This may occur as an acute,

transient condition in severe exertion in an untrained or feeble condition—during, for example, the ascent of a mountain.

There may be great dilatation of the right heart, as shown by the increased epigastric pulsation, and even increase in the cardiac dulness. The safety valve action of the tricuspid valves may here come into play, relieving the lungs by permitting regurgitation into the auricle. With rest the condition is removed, but, if it has been extreme, the heart may suffer a strain from which it may recover slowly, or, indeed, the individual may never be able again to undertake severe exertion. In the process of training the getting wind, as it is called, is largely a gradual increase in the capability of the heart, particularly of the right chambers. A degree of exertion can be safely maintained in full training which would be quite impossible under other circumstances, because, by a gradual process of what we may call physical education, the heart has strengthened its reserve force—widened enormously its limit of physiological work. Endurance in prolonged contests is measured by the capabilities of the heart, which by increasing its tonus has increased its resistance to dilatation. We have no positive knowledge of the nature of the changes in the heart which occur in this process, but it must be in the direction of increased muscular and nervous energy. The large heart of athletes may be due to the prolonged use of their muscles, but no man becomes a great runner or oarsman who has not naturally a capable if not a large heart. Master McGrath, the celebrated greyhound, and Eclipse, the race horse, both famous for endurance rather than speed, had very large hearts.

Excessive dilatation during severe muscular effort results in *heart-strain*. A man, perhaps in poor condition, calls upon his heart for extra work during the ascent of a high mountain, and is at once seized with pain about the heart and a sense of distress in the epigastrium. He breathes rapidly for some time, is "puffed," as we say, but the symptoms pass off after a night's quiet. An attempt to repeat the exercise is followed by another attack, or, indeed, an attack of cardiac dyspnœa may come on while he is at rest. For months such a man may be unfitted for severe exertion, or he may be permanently incapacitated. In some way he has overstrained his heart and become "broken-winded." Exactly what has taken place in these hearts we can not say, but their reserve force is lost, and with it the power of meeting the demands exacted in maintaining the circulation during severe exertion. The "heart-shock" of Latham includes cases of this nature—sudden cardiac breakdown during exertion, not due to rupture of a valve. It seems probable that sudden death in men during long continued efforts, as in a race, is sometimes due to overdistention and paralysis of the heart.

Acute dilatative heart weakness is seen in many conditions, as in Graves' disease, in paroxysmal tachycardia, in old myocardial cases following exertion, and in angina pectoris. There is usually a striking contrast between the wide and forcible cardiac impulse and the small, feeble, irregular pulse.

Dilatation occurs in all forms of *valve lesions*. In aortic incompetency blood enters the left ventricle during diastole from the unguarded aorta and from the left auricle, and the quantity of blood at the termination of diastole subjects the walls to an extreme degree of pressure, under which they inevitably yield. In time they augment in thickness, and present the typical eccentric hypertrophy of this condition.

In mitral insufficiency blood which should have been driven into the aorta is forced into and dilates the auricle from which it came, and then in the diastole of the ventricle a large amount is returned from the auricle, and with increased force. In mitral stenosis the left auricle is the seat of greatly increased tension during diastole, and dilates as well as hypertrophies; the distention, too, may be enormous. Dilatation of the right ventricle is produced by a number of conditions, which were considered under hypertrophy. All circumstances, such as mitral stenosis, emphysema, etc., which permanently increase the tension of the blood in the pulmonary vessels cause its dilatation.

The idiopathic dilatation and hypertrophy of beer drinkers also comes in this group, as it is brought about gradually by increased endocardial pressure.

(*b*) IMPAIRED NUTRITION OF THE HEART WALLS may lead to a diminution of the resisting power so that dilatation readily occurs.

The loss of tone due to parenchymatous degeneration or myocarditis in fevers may lead to a fatal condition of acute dilatation. It is a recognized cause of death in scarlatinal dropsy (Goodhart), and may occur in rheumatic fever, typhus, typhoid, erysipelas, etc. The changes in the heart muscle which accompany acute endocarditis or pericarditis may lead to dilatation, especially in the latter disease. In anæmia, leukæmia, and chlorosis the dilatation may be considerable. In sclerosis of the walls the yielding is always where this process is most advanced, as at the left apex. Under any of these circumstances the walls may yield with normal blood pressure.

Pericardial adhesions are a cause of dilatation, and we generally find in cases with extensive and firm union considerable hypertrophy and dilatation. There is usually here some impairment as well of the superficial layers of muscle.

3. CARDIAC INSUFFICIENCY

Etiology.—With lessening of the muscular power of the heart the rapidity with which the blood circulates is diminished, and the tissues fail to receive their proper supply of oxygen and food, and to be adequately relieved of their waste products—this is cardiac failure. The same effect may be produced in another way. The amount of blood in the body is much less than the total capacity of the vascular bed, and an adequate blood supply is only kept up by a general constriction of arterioles which dam the blood in the arterial system, but if by any chance there is a general vaso-dilatation of the arterioles, especially those in the splanchnic area, the heart does not receive an amount of blood sufficient to supply the bodily needs, with the same effect on the organs as in certain forms of cardiac failure. This condition, which is probably the essence of shock, does not concern us here, but it must be mentioned to avoid the impression that all failure of the circulation means failure of the heart.

The failure in muscular power may affect any cavity singly or the whole heart. Weakness of the left ventricle fails to give proper filling of the arterial system and general anæmia of the tissues results. Failure of the left auricle means stasis in the lung vessels with deficient aeration of the blood, and a tendency to œdema of the lung or to effusion into the pleural cavity.

Failure of the right auricle and ventricle gives cyanosis of the organs, dyspnœa at rest and on slight exertion, with stasis in the abdominal organs and œdema.

The reserve power with which the cardiac muscle is endowed disappears in heart failure. This reserve, greatest in youth, is increased by adequate nutrition, certain congenital endowments, and, apart from other defects, by hypertrophy. It is lessened by defects in the cardiac structure, gross or minute, by defective nutrition, by certain bacterial and other poisons, and with advancing years. We have at present no means of gauging this reserve power of the organ as a whole or in its different parts.

The failure may be sudden or slow, according to the kind and rapidity of the lesion which causes it. When the left ventricle fails the effect may vary from immediate death, through all forms of fainting, giddiness, sense of dissolution, to a mild sense of bodily or mental fatigue; when the right ventricle fails the effect varies from a sudden dyspnœa to a dyspnœa which comes on with slight exertion.

As to the actual condition in cardiac failure generally, it is by no means easy in all cases to say what has been the cause. The lesions to which the cardiac musculature is liable are described further on, yet there is a proportion of cases in which neither by post mortem examination nor careful microscopic search can the source of the failure be even suggested. It is well to bear in mind a suggestion which has been made by Aschoff, namely, that in certain cases the failure is due not so much to the implication of the general musculature as to an affection of the conducting muscular system of Tawara's node in the inter-auricular septum, and of the bundle of His with its ramifications which stretch to all parts of the right and left ventricles, and whose function is to distribute the muscular impulses which at each contraction spread from the venous to the arterial end of the heart.

The blood pressure in cardiac insufficiency shows no uniform figures. The maximum pressure, which is that usually estimated, may be high even in a failing heart. In serious degrees of myocardial affection, such as fatty degeneration or chloroform poisoning, it is low. In cases in which there has been a raised blood pressure, the maximum may be lower or higher than the normal for the patient. We must recognize that probably in early stages of failure the heart is stimulated to put forth increased energy at each beat, and that the maximum pressure at the height of the beat slightly over-compensates the circulatory defect.

Acute Cardiac Insufficiency.—Causes: (*a*) Wounds of the heart, (*b*) spontaneous rupture or rupture of valves, (*c*) rapid effusion into the pericardium of blood or serous fluid, (*d*) access of air to the chambers of the heart, as from operations at the root of the neck or decomposition after exposure to a high atmospheric pressure, (*e*) large thrombi quickly formed in a heart cavity, (*f*) sudden interference with the coronary circulation, especially the left coronary artery, (*g*) mechanical interference with the heart from obliteration of the trachea or larynx, as in strangulation, (*h*) acute infections, such as diphtheria or pericarditis, (*i*) certain poisons, such as pilocarpin, cocaine, phosphorus, etc., (*j*) stimulation of the vagus nerve, its centre in the medulla, or its termination in the heart.

Chronic Cardiac Insufficiency.—Causes: (*a*) Lesions of the heart muscle, which will be described in more detail in a subsequent paragraph.

Essentially all cardiac failure is muscular. The myocardium may be insufficiently nourished, as in the starvation atrophy of new growths or in diabetes, or there may be recognizable gross or microscopic lesions. One or more of the functions of the cardiac muscle may be interfered with without producing any changes that can be detected by the microscope, such as the failure associated with aortic disease. (*b*) Lesions of the valves. (*c*) Lesions affecting the vascular fields of the efferent arteries. Emphysema, chronic bronchitis, asthma, sclerosis of the lungs, chest deformities, and mitral disease produce an embarrassment of the right heart; atheroma of the aorta and arterio-sclerosis, especially of the splanchnic and renal area, produce failure of the left heart. (*d*) Over-exertion. (*e*) Certain poisons, such as alcohol (especially beer) and phosphorus. (*f*) Other causes, such as adherent pericardium and exophthalmic goitre.

Anatomical Basis of Cardiac Insufficiency.—1. Lesions Due to Disease of the Coronary Arteries.—A knowledge of the changes produced in the myocardium by disease of the coronary vessels gives a key to the understanding of many problems in cardiac pathology. The terminal branches of the coronary vessels are end arteries; that is, the communication between neighboring branches is through capillaries only. J. H. Pratt has shown that the vessels of Thebesius, which open from the ventricles and auricles into a system of fine branches and thus communicate with the cardiac capillaries and coronary veins, may be capable of feeding the myocardium sufficiently to keep it alive even when the coronary arteries are occluded. The blocking of one of these vessels by a thrombus or an embolus leads usually to ~~a condition which is known as~~

(*a*) *Anæmic necrosis,* or white infarct. When this does not occur the reason may be sought in (1) the existence of abnormal anastomoses, which by their presence take the coronary system out of the group of end arteries; or (2) the vicarious flow through the vessels of Thebesius and the coronary veins. The condition is most commonly seen in the left ventricle and in the septum, in the territory of distribution of the anterior coronary artery. The affected area has a yellowish white color, sometimes a turbid, parboiled aspect, at other times a grayish red tint. It may be somewhat wedge-shaped, more often it is irregular in contour and projects above the surface. Microscopically the changes are very characteristic. The nuclei either disappear from the muscle fibres or they undergo fragmentation. Leucocytes wander in from the surrounding tissue, and these may suffer disintegration. At a later stage a new growth of fibrous tissue is found in the periphery of the infarct which ultimately may entirely replace the dead fibres. The fibres present a homogeneous, hyaline appearance. In some instances there is complete transformation, and even to the naked eye a firm white patch of hyaline degeneration may appear in the centre of the area. Rupture of the heart may be associated with anæmic necrosis.

(*b*) The second important effect of coronary-artery disease upon the myocardium is seen in the production of *fibrous myocarditis.* This may result from the gradual transformation of areas of anæmic necrosis. More commonly it is caused by the narrowing of a coronary branch in a process of obliterative endarteritis. Where the process is gradual evidences of granulation tissue are often wanting, and any distinction between the necrotic muscle

fibres and the new scar tissue is difficult to establish. J. B. MacCallum showed that the muscle fibres undergo a change the reverse of that of their normal development and lose their fibril bundles preliminary to their complete replacement by connective tissue. The sclerosis is most frequently seen at the apex of the left ventricle and in the septum, but it may occur in any portion. In the septum and walls there are often streaks and patches which are only seen in carefully made serial sections. Hypertrophy of the heart is commonly associated with this degeneration. It is the invariable precursor of aneurism of the heart.

(*c*) *Sudden Death in Coronary Artery Disease.*—Complete obliteration of one coronary artery, if produced suddenly, is usually fatal. When induced slowly, either by arterio-sclerosis at the orifice of the artery at the root of the aorta or by an obliterating endarteritis in the course of the vessel, the circulation may be carried on through the other vessel. Sudden death is not uncommon, owing to thrombosis of a vessel which has become narrowed by sclerosis. *In medico-legal cases it is a point of primary importance to remember that this is one of the common causes of sudden death.* This condition should be carefully sought for, inasmuch as it may be the sole lesion, except a general, sometimes slight, arterio-sclerosis. In the most extreme grade one coronary artery may be entirely blocked, with the production of extensive fibroid disease, and a main branch of the other also may be occluded. A large, powerfully built imbecile, aged thirty five, at the Elwyn Institution, Pennsylvania, who had for years enjoyed doing the heavy work about the place, died suddenly, without any preliminary symptoms. The heart weighed over 600 grams; the anterior coronary artery was practically occluded by obliterating endarteritis, and of the posterior artery one main branch was blocked.

(*d*) *Septic Infarcts.*—In pyæmia the smaller branches of the coronary arteries may be blocked with emboli which give rise to infectious or septic infarcts in the myocardium in the form of abscesses, varying in size from a pea to a pin's head. These may not cause any disturbance, but when large they may perforate into the ventricle or into the pericardium, forming what has been called acute ulcer of the heart.

II. Acute Interstitial Myocarditis.—In some infectious diseases and in acute pericarditis the intermuscular connective tissue may be swollen and infiltrated with small round cells and leucocytes, the blood vessels dilated, and the muscle fibres the seat of granular, fatty, and hyaline degeneration. Occasionally, in pyæmia the infiltration with pus cells has been diffuse and confined chiefly to the interstitial tissue. Councilman has described this condition of the heart wall in gonorrhœa, and succeeded in demonstrating the gonococcus in the diseased areas. The commonest examples are found in diphtheria, typhoid fever, and acute endocarditis, as shown by the studies of Romberg. The foci may be the starting points of patches of fibrous myocarditis.

III. Fragmentation and Segmentation.—This condition was described by Renaut and Landouzy in 1877, and has been carefully studied by different pathologists. Two forms are met with: 1. Segmentation. The muscle fibres have separated at the cement line. 2. Fragmentation. The fracture has been across the fibre itself, and perhaps at the level of the nucleus. Longitudinal division is unusual. Although the condition doubtless arises in some instances during the death agony, as in cases of sudden death by vio-

lence, in others it would seem to have clinical and pathological significance. It is found associated with other lesions, fibrous myocarditis, infarction, and fatty degeneration. J. B. MacCallum distinguished a simple from a degenerative fragmentation. The first takes place in the normal fibre, which, however, shows irregular extensions and contractions. The second succeeds degeneration in the fibre. Hearts the seat of marked fragmentation are lax, easily torn, the muscle fibres widely separated, and often pale and cloudy.

IV. Parenchymatous Degeneration.—This is usually met with in fevers, or in connection with endocarditis or pericarditis, and in infections and intoxications generally. It is characterized by a pale, turbid state of the cardiac muscle, which is general, not localized. Turbidity and softness are the special features. It is the softened heart of Laennec and Louis. Stokes speaks of an instance in which "so great was the softening of the organ that when the heart was grasped by the great vessels and held with the apex pointing upward, it fell down over the hand, covering it like a cap of a large mushroom."

Histologically, there is a degeneration of the muscle fibres, which are infiltrated to a various extent with granules which resist the action of ether, but are dissolved in acetic acid. Sometimes this granular change in the fibres is extreme, and no trace of the striæ can be detected. It is probably the effect of a toxic agent, and is seen in its most exquisite form in the lumbar muscles in cases of toxic hæmoglobinuria in the horse. It is met with in cases of typhoid, typhus, small-pox, and other infectious diseases, particularly when the course is protracted. There is no definite relation between it and the high temperature.

V. Fatty Heart.—Under this term are embraced fatty degeneration and fatty overgrowth.

(*a*) *Fatty degeneration* is a very common condition, and mild grades are met with in many diseases. It is found in the failing nutrition of old age, of wasting diseases, and of cachectic states; in prolonged infectious fevers, in which it may follow or accompany the parenchymatous change. In pernicious anæmia and in phosphorus poisoning the most extreme degrees are seen. Pericarditis is usually associated with fatty or parenchymatous changes in the superficial layers of the myocardium. Disease of the coronary arteries is a much more common cause of fibroid degeneration than of fatty heart. Lastly, in the hypertrophied ventricular wall in chronic heart-disease fatty change is by no means infrequent. This degeneration may be limited to the heart or it may be more or less general in the solid viscera. The diaphragm may also be involved, even when the other muscles show no special changes. There appears to be a special proneness to fatty degeneration in the heart muscle, which may ~~perhaps~~ be connected with its incessant activity. So great is its need of an abundant oxygen supply that it feels at once any deficiency, and is in consequence the first muscle to show nutritional changes.

Anatomically the condition may be local or general. The left ventricle is most frequently affected. If the process is advanced and general, the heart looks large and is flabby and relaxed. It has a light yellowish brown tint, or, as it is called, a faded leaf color. Its consistence is reduced and the substance tears easily. In the left ventricle the papillary columns and the muscle beneath the endocardium show a streaked or patchy appearance. Microscop-

ically, the fibres are seen to be occupied by minute globules distributed in rows along the line of the primitive fibres (Welch). In advanced grades the fibres seem completely occupied by the minute globules.

(*b*) *Fatty Overgrowth.*—This is usually a simple excess of the normal subpericardial fat, to which the term *cor adiposum* was given by the older writers. In pronounced instances the fat infiltrates between the muscular substance and, separating the strands, may reach even to the endocardium. In corpulent persons there is always much pericardial fat. It forms part of the general obesity, and occasionally leads to dangerous or even fatal impairment of the contractile power of the heart. Of 122 cases analyzed by Forchheimer there were 88 males and 34 females. Over 80 per cent. occurred between the fortieth and seventieth years.

The entire heart may be enveloped in a thick sheeting of fat through which not a trace of muscle substance can be seen. On section the fat infiltrates the muscle, separating the fibres, and in extreme cases—particularly in the right ventricle—reaches the endocardium. In some places there may be even complete substitution of fat for the muscle substance. In rare instances the fat may be in the papillary muscles. The heart is usually much relaxed and the chambers are dilated. Microscopically the muscle fibres may show, in addition to the atrophy, marked fatty degeneration.

VI. Other Degenerations of the Myocardium.—(*a*) *Brown Atrophy.*—This is a common change in the heart muscle, particularly in chronic valvular lesions and in the senile heart. When advanced the color of the muscles is a dark red brown, and the consistence is usually increased. The fibres present an accumulation of yellow brown pigment chiefly about the nuclei. The cement substance is often unusually distinct, but seems more fragile than in healthy muscle.

(*b*) *Amyloid degeneration of the heart* is occasionally seen. It occurs in the intermuscular connective tissue and in the blood vessels, not in the fibres.

(*c*) The *hyaline transformation of Zenker* is sometimes met with in prolonged fevers. The affected fibres are swollen, homogeneous, translucent, and the striæ are very faint.

(*d*) *Calcareous degeneration* occasionally occurs in the myocardium, and the muscle fibres may be infiltrated with lime salts and yet retain their appearance.

Symptoms of Cardiac Insufficiency.—As indicated above, the symptoms of left sided cardiac failure differ from those of the right side, and in each we may distinguish a number of types, which, however, merge gradually the one into the other. Failure of the left ventricle is seen in its severest forms in the abrupt death stroke of angina pectoris, in the sudden faints with sweats and heart pain of fatty or fibroid hearts, or in the fainting and convulsive attacks of Stokes-Adams disease. Less severe failure may be seen in athletes after a hard race, when vomiting and a feeling of dissolution are present—a type which is sometimes seen in angina, when it is liable to be mistaken for a gastro-intestinal upset. The milder degrees show themselves in an inability to take much exercise or to suffer much mental work without the sense of great fatigue. Sudden and slow types are also seen in failure of the right side. Subjected to a slight strain, great hyperpnœa and distress may come on, and one form of cardiac dyspnœa which attacks the patient at night is

of this nature. The severer forms show an increasing inability to undergo slight extra exertion, such as mounting stairs, or hyperpnœa even when at rest in bed, in both of which there is usually some œdema of the feet, especially at night, if the patient is on his feet most of the day.

Grouped under their special systems the symptoms complained of by patients with cardiac failure are as follows: (*a*) Cardio-vascular system: Pain in the cardiac area or extending to the shoulders and down the arms, a sense of weight in the præcordium; palpitation is seldom complained of. (*b*) Respiratory system: Dyspnœa at rest or on exertion, or orthopnœa, Cheyne-Stokes respiration, cough, loss of voice from pressure of a dilated left auricle on the left recurrent laryngeal nerve, hæmoptysis (as resulting from lung infarcts). (*c*) Central nervous system; in addition to those given above are sleeplessness, mental symptoms, delusions, melancholia, and especially toward the end stupor and drowsiness. (*d*) Cyanosis, pallor, œdema, and occasionally purpura in the lower limbs. (*e*) Alimentary system: The stasis in the abdominal organs in right heart failure produces loss of appetite, indigestion, flatulence, vomiting, constipation, diarrhœa, abdominal pain, hæmorrhoids, etc. (*f*) Renal system: The urine is scanty, high colored, and contains a slight amount of albumin.

Physical examination of the heart may reveal an apex-beat which is feeble, outside the nipple line, diffuse, and whose maximum intensity is not easily localized. The pulsation may be marked on inspection and cover a very wide area; arterial pulsation in the neck in the left heart failure may be great; in right heart failure the jugular veins may be very dilated and their pulsations may be normal, indicating three waves, or regurgitant. On percussion the cardiac area may be much increased to the right or to the left, or both. On auscultation the sounds may be difficult to hear, or feebler than normal; murmurs, usually soft, may be present at both apex and base. Gallop rhythm may be present. The pulse may show great variations; marked failure may exist with a full bounding pulse; more usually it is feeble with diminished tension; it may be irregular, intermittent, slow, or rapid. No one sign or combination of signs is significant of cardiac failure. A heart may be insufficient and yet perhaps nothing can be detected by physical examination except feeble sounds and a low tension pulse.

The myocardial lesion is not always proportionate to the intensity of the symptoms. A patient may present enfeebled, irregular action and signs of dilatation—shortness of breath, œdema, and the general symptoms believed to be characteristic of cases of fibroid and fatty heart—and the post mortem show little or no change in the myocardium.

Cardio-sclerosis or fibroid heart is in some cases characterized by a feeble, irregular, slow pulse, with dyspnœa on exertion and occasional attacks of angina. Irregularity is present in many, but not in all cases. The pulse may be very slow, even 30 or 40 per minute, and the features those of Stokes-Adams disease. A man with advanced fibroid myocarditis may die suddenly while at work, without having ever complained of heart trouble. Ultimately the cases come under observation with the symptoms of cardiac insufficiency. The arrhythmia, which may have been present, becomes aggravated and, according to Riegel, may not only precede, but also persist after the cardiac insufficiency has passed away.

Dyspnoea. - Dwelō - Decrease in vital capacity of lungs
hyperpnoea -
[illegible]
[illegible]

Test of functional capacity. Katzenstein's method

Pulse b.p. on rising several times. Then told [illegible]
[illegible] blood pressure taken for 2½ minutes. Then pulse & bp
[illegible] - If heart capable - b.p. rises but pulse same or slower
weak heart. = " does not rise but [illegible] pulse
becomes more [illegible]

"Drop Heart". Subjective symptoms common much like those of
[illegible] new. dyspn- ankles [illegible] urine scanty. Veins [illegible]
[illegible] by gen. condit. change by rest + digitalis, + [illegible]
Effect by rest + digitalis is said to [illegible]

Fatty degeneration of the heart presents the same difficulties. Extreme fatty changes, as in pernicious anæmia, may be present with a full pulse and regularly acting heart. The fat does not appear to interfere seriously with the function of the organ. The truth is, it may be present in an extreme grade without producing symptoms, so long as great dilatation of the chambers does not occur. The cardiac irregularity, the dyspnœa, palpitation and small pulse are in reality not symptoms of the fatty degeneration, but of dilatation which has supervened. The fatty *arcus senilis* is of no moment in the diagnosis of fatty heart. The heart sounds may be weak and the action irregular.

When dilatation occurs there is gallop rhythm, shortening of the long pause, and a systolic murmur at the apex. Shortness of breath on exertion is an early feature in many cases, and anginal attacks may occur. There is sometimes a tendency to syncope, and in both fibroid and fatty heart there are attacks in which the patient feels cold and depressed and the pulse sinks to 40 or 30, or even, as in one case which I saw, to 26. The patient may wake from sleep in the early morning with an attack of severe "cardiac asthma." These "spells" may be associated with nausea and may alternate with others in which there are anginal symptoms. These are the cases, too, in which for weeks there may be mental symptoms. The patient has delusions and may even become maniacal. Toward the close the type of breathing known as Cheyne-Stokes may occur. It was described in the following terms by John Cheyne, speaking of a case of fatty heart (Dublin Hospital Reports, vol. ii, p. 221, 1818): "For several days his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again: this revolution in the state of his breathing lasted about a minute, during which there were about thirty acts of respiration." It is seen much more frequently in arterio-sclerosis and uræmic states than in fatty heart.

Fatty overgrowth of the heart is a condition certain to exist in very obese persons. It produces no symptoms until the muscular fibre is so weakened that dilatation occurs. These patients may for years present a feeble but regular pulse; the heart sounds are weak and muffled, and a murmur may be heard at the apex. Attacks of "cardiac asthma" are not uncommon, and the patient may suffer from bronchitis. Dizziness and pseudo-apoplectic seizures may occur. Sudden death may result from syncope or from rupture of the heart. The physical examination is often difficult because of the great increase in the fat, and it may be impossible to define the area of dulness.

For clinical purposes we may group the cases of failure from myocardial disease as follows:

(1) Those in which sudden death occurs with or without previous indications of heart-trouble. Sclerosis of the coronary arteries exists—in some instances with recent thrombus and white infarcts; in others, extensive fibroid disease; in others again, fatty degeneration. Many patients never complain of cardiac distress, but, as in the case of Chalmers, the celebrated Scottish divine, enjoy unusual vigor of mind and body.

(2) Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of dyspnœa, sometimes anginal attacks, collapse symptoms

with sweats and extremely slow pulse, and occasionally marked mental symptoms.

(3) Cases with general arterio-sclerosis and hypertrophy and dilatation of the heart. They are robust men of middle age who have worked hard and lived carelessly Dyspnœa, cough, and swelling of the feet are the early symptoms, and the patient comes under observation either with a gallop rhythm, embryocardia, or an irregular heart with an apex systolic murmur of mitral insufficiency. Recovery from the first or second attack is the rule. It is one of the most common forms of heart-disease.

Prognosis.—Each case must be judged on its own merits, special notice being taken of the age, probable origin, and anatomical basis of the insufficiency. The outlook in affections of the myocardium occurring late in life is extremely grave. Patients recover, however, in a surprising way from the most serious attacks, particularly those of the third group.

Treatment.—Many cases never come under treatment; the first are the final symptoms. Other cases with well marked failure, if treated on general lines according to the routine measures, recover quickly.

Much more difficult is the management of those cases in which there is marked cardiac arrhythmia, with a feeble, irregular, very slow pulse, and syncope or angina. Dropsy is not, as a rule, present; the heart sounds may be clear and there are no signs of dilatation.

The following are the general methods to be observed in the treatment of cardiac failure:

(*a*) REST.—Disturbed compensation may be completely restored by rest of the body. In many cases with œdema of the ankles, moderate dilatation of the heart, and irregularity of the pulse, rest in bed, a few doses of the compound tincture of cardamon, and a saline purge suffice, within a week or ten days, to restore the compensation.

(*b*) DIET.—In acute conditions it is usually well to limit this in amount, especially the fluids. With marked passive congestion liquid diet may be advisable; otherwise small amounts of simple food may be given at short intervals. In any case with dilatation it is well to limit the total daily intake of fluids to 1,500 c. c. A "dry diet" for a few days is sometimes useful.

(*c*) THE RELIEF OF THE EMBARRASSED CIRCULATION.

(1) *By Venesection.*—In cases of dilatation, from whatever cause, in mitral or aortic lesions or distention of the right ventricle in emphysema, when signs of venous engorgement are marked and when there is orthopnœa with cyanosis, the abstraction of from 20 to 30 ounces of blood is indicated. This is the occasion in which timely venesection may save the patient's life. It is particularly helpful in the dilated heart of arterio-sclerosis.

(2) *By Depletion through the Bowels.*—This is particularly valuable when dropsy is present. Of the various purges the salines are to be preferred and may be given by Matthew Hay's method. Half an hour to an hour before breakfast from half an ounce to an ounce and a half of Epsom salts may be given in a concentrated form. This usually produces from three to five liquid evacuations. The compound jalap powder in half drachm doses, or elaterium, may be employed for the same purpose. Even when the pulse is very feeble these hydragogue cathartics are well borne, and they deplete the portal system rapidly and efficiently.

(3) *The Use of Remedies Which Stimulate the Heart's Action.*—Of these by far the most important is digitalis, which was introduced into practice by Withering. The indication for its use is weakness of the heart muscle: the contra-indication is a perfectly balanced compensatory hypertrophy, such as we see in all forms of valvular disease. Broken compensation in valvular disease, no matter what the lesion may be, is the signal for its use. It acts upon the heart, slowing and at the same time increasing the force of the contractions. It acts on the peripheral arteries, raising their tension, so that a steady and equable flow of blood is maintained in the capillaries, which, after all, is the prime aim and object of the circulation. High blood pressure is not a contra-indication to its use. The beneficial effects are best seen in cases of mitral disease with small, irregular pulse and cardiac dropsy. Its effects are not less striking in the dilatation of the left ventricle, in the failing compensation of aortic insufficiency or of arterio-sclerosis. On theoretical grounds it has been urged that its use is not so advantageous in aortic insufficiency, since it prolongs the diastole and leads to greater distention. This need not be considered, and digitalis is just as serviceable in this as in any other condition associated with progressive dilatation; larger doses are often required. It may be given as the tincture or the infusion. In cases of cardiac dropsy, from whatever cause, 15 minims (1 c. c.) of the tincture or half an ounce (15 c. c.) of the infusion may be given every three hours for two days, after which the dose may be reduced. Some prefer the tincture, others the infusion; it is a matter of indifference if the drug is good. The urine of a patient taking digitalis should be carefully estimated each day. As a rule, when its action is beneficial, there is within twenty-four hours an increase in the amount; often the flow is very great. Under its use the dyspnœa is relieved, the dropsy gradually disappears, the pulse becomes firmer, fuller in volume, and sometimes, if it has been very intermittent, regular.

Ill effects sometimes follow digitalis. There is no such thing as a cumulative action of the drug manifested by sudden symptoms. Toxic effects are seen in the production of nausea and vomiting. Digipuratum is less disturbing to the stomach and may be given when there is gastric irritability. The pulse becomes irregular and small, and there may be two beats of the heart to one of the pulse, which, as pointed out by Broadbent, is found particularly in cases of mitral stenosis when they are under the influence of this drug. The urine is reduced in amount. These symptoms subside on the withdrawal of the digitalis, and are rarely serious. There are patients who take digitalis uninterruptedly for years, and feel palpitation and distress if the drug is omitted. In mitral disease, even when it does good, it does not always steady the pulse. There are many cases in which the irregularity is not affected by the digitalis. When the compensation has been re-established the drug may be omitted. When there is dyspnœa on exertion and cardiac distress, from 5 to 10 minims (0.3 to 0.6 c. c.) three times a day may be advantageously given for prolonged periods, but the effects should be carefully watched. In cardiac dropsy digitalis should be used at the outset with a free hand. Small doses should not be given, but from the first half-ounce doses of the infusion every three hours, or from 15 to 20 minims of the tincture. Digitalin, hypodermically (gr. 1/30, 0.002 gm.), every three or four hours, may be substituted.

Of other remedies strophanthus alone is of service, but as its effect is uncertain when given by mouth it should be administered by intramuscular injection. Doses of 10 to 15 minims (0.6 to 1 c.c.) of the tincture or strophanthin gr. 1/100 (0.00065 gm.) are given and repeated once or twice after intervals of twenty-four hours. The intramuscular is safer than the intravenous administration. It certainly will sometimes steady the intermittent heart of mitral valve disease when digitalis fails to do so. Convallaria, ~~citrate of~~ caffeine, ~~and~~ adonis vernalis and sparteine are ~~usually~~ recommended as substitutes for digitalis, but their inferiority is so manifest that their use is rarely indicated.

There are two valuable adjuncts in the treatment of valvular disease—iron and strychnia. When anæmia is a marked feature iron should be given in full doses. In some instances of failing compensation this is the only medicine needed to restore the balance. Arsenic is occasionally an excellent substitute, and one or other of them should be administered in all instances of heart trouble when pallor is present. Strychnia is a heart tonic of very great value. It may be given alone or in combination with the digitalis in 1 or 2 drop doses of the 1 per cent. solution, or hypodermically in doses of 1/40-1/10 gr. (0.0016 to 0.006 gm.). ~~Alcoholic stimulants in moderation are occasionally useful, especially in tiding over a period of acute cardiac weakness.~~

Treatment of Special Symptoms.—(*a*) Dropsy.—The increased arterial tension and activity of the capillary circulation under the influence of digitalis hasten the interstitial lymph flow and favor resorption of the fluid. The hydragogue cathartics, by rapidly depleting the blood, promote, too, the absorption of the fluid from the lymph spaces and the lymph sacs. These two measures usually suffice to rid the patient of the dropsy. In some cases, however, it can not be relieved, and then ~~Southey's tubes may be used or~~ the legs may be punctured by ordinary aspirating needles with rubber tubing attached, which may be inserted and left for hours; they often drain away large amounts. If done with care, after a thorough washing of the parts, and if antiseptic precautions are taken, scarification is a ~~very~~ serviceable measure, ~~and should be resorted to more frequently than it is.~~ Canton flannel bandages may be applied on the œdematous legs. In case of marked hydrothorax or ascites tapping is advisable before digitalis is given.

(*b*) Dyspnœa.—The patients are usually unable to lie down. A comfortable bed-rest should therefore be provided—if possible, one with lateral projections, so that in sleeping the head can be supported as it falls over. The shortness of breath is associated with dilatation, chronic bronchitis, or hydrothorax. The chest should be carefully examined in all these cases, as hydrothorax of one side or of both is a common cause of shortness of breath. There are cases of mitral regurgitation with recurring hydrothorax usually on the right side, which is relieved, week by week or month by month, by tapping. For the nocturnal dyspnœa, particularly when combined with restlessness, morphia is invaluable and may be given without hesitation. The value of the calming influence of opium in all conditions of cardiac insufficiency is not sufficiently recognized. There are instances of cardiac dyspnœa unassociated with dropsy, particularly in mitral valve disease, in which nitroglycerin is of great service, if given in the 1 per cent. solution in increasing doses. It is especially serviceable in the cases in which the pulse tension is high.

(*c*) Palpitation and Cardiac Distress.—In instances of great hypertrophy and in the throbbing which is so distressing in some cases of aortic insufficiency, aconite is of service in doses of from 1 to 3 drops every two or three hours. An ice bag over the heart ~~or Leiter's coil~~ is also of service in allaying the rapid action and the throbbing. For the pains, which are often so marked in aortic lesions, iodide of potassium in 10-grain (0.6 gm.) doses, three times a day, or nitroglycerin may be tried. Small blisters are sometimes advantageous. It must be remembered that an important cause of palpitation and cardiac distress is flatulent distention of the stomach or colon, against which suitable measures must be directed.

(*d*) Gastric Symptoms.—The cases of cardiac insufficiency which do badly and fail to respond to digitalis are most often those in which nausea and vomiting are prominent features. The liver is often greatly enlarged in these cases; there is more or less stasis in the hepatic vessels, and but little can be expected of drugs until the venous engorgement is relieved. If the vomiting persists, it is best to stop the food and give small bits of ice, small quantities of milk and lime water, and effervescing drinks, ~~such as Apollinaris water and champagne.~~ The bowels should be freely moved and drugs given hypodermically, if possible.

(*e*) Cough and Hæmoptysis.—The former is almost a necessary concomitant of cardiac insufficiency, owing to engorgement of the pulmonary vessels and more or less bronchitis. It is allayed by measures directed rather to the heart than to the lungs. Hæmoptysis in chronic valvular disease is sometimes a salutary symptom. An army surgeon, who was invalided during the American civil war on account of hæmoptysis, supposed to be due to tuberculosis, had for many years, in association with mitral insufficiency and enlarged heart, many attacks of hæmoptysis. He assured me that his condition was invariably better after the attack. It is rarely fatal, except in some cases of acute dilatation, and seldom calls for special treatment.

(*f*) Sleeplessness.—One of the most distressing features, ~~of valvular lesions~~, even in the stage of compensation, is disturbed sleep. Patients may wake suddenly with throbbing of the heart, often in an attack of nightmare. Subsequently, when the compensation has failed, it is also a worrying symptom. The sleep is broken, restless, and frequently disturbed by frightful dreams. Sometimes a dose of the spirit of chloroform or of ether, with half a drachm of spirit of camphor, given in a little hot whisky, will give a quiet night. The compound spirit of ether, Hoffmann's anodyne, though very unpleasant to take, is frequently a great boon in the intermediate period when compensation has partially failed and the patients suffer from restless and sleepless nights. Paraldehyde and amylene hydrate are sometimes serviceable, but it is best, after a few trials, particularly if the paraldehyde does not answer, to resort to morphia. It may be given in combination with atropine.

(*g*) Renal Symptoms.—With broken compensation and lowering of the tension in the aorta, the urinary secretion is greatly diminished, and the amount may sink to 5 or 6 ounces in the day. Digitalis and strophanthus usually increase the flow. A brisk purge may be followed by augmented secretion. The combination in pill form of digitalis, squill, and calomel will sometimes prove effective when the infusion or tincture of digitalis alone has

failed. Calomel acts well in some cases, given in 3 grain (0.2 gm.) doses every six hours for three or four days.

The DIET in chronic valvular disease is often very difficult to regulate. Widal and others have shown that retention of the chlorides is an important factor in cardiac dropsy and heart failure. A milk diet, 2 litres a day, favors their elimination, and in the intervals between attacks a salt free diet as far as possible should be used. Starchy foods and all articles likely to cause flatulency should be forbidden. Stimulants are often necessary, either whisky or brandy.

In certain cases of weak heart, particularly when it is due to fatty overgrowth, the plans recommended by Oertel and by Schott are advantageous. They are invaluable methods in those forms of heart weakness due to intemperance in eating and drinking and defective bodily exercise. The Oertel plan consists of three parts: First, the reduction in the amount of liquid. This is an important factor in reducing the fat in these patients. It also slightly increases the density of the blood. Oertel allows daily about 36 ounces of liquid, which includes the amount taken with the solid food. Free perspiration is promoted by bathing (if advisable, the Turkish bath), or even by the use of pilocarpine.

The second important point in his treatment is the diet, which should consist largely of proteids.

Morning.—Cup of coffee or tea, with a little milk, about 6 ounces altogether. Bread, 3 ounces.

Noon.—Three to 4 ounces of soup, 7 to 8 ounces of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; 1 ounce of bread or farinaceous pudding; 3 to 6 ounces of fruit for dessert. No liquids at this meal, as a rule, but in hot weather 6 ounces of light wine may be taken.

Afternoon.—Six ounces of coffee or tea, with as much water. As an indulgence an ounce of bread.

Evening.—One or 2 soft-boiled eggs, an ounce of bread, perhaps a small slice of cheese, salad, and fruit; 6 to 8 ounces of wine with 4 or 5 ounces of water (Yeo).

The most important element of all is graduated exercise, not on the level, but up hills of various grades. The distance walked each day is marked off and is gradually lengthened. In this way the heart is systematically exercised and strengthened.

The Schott Treatment.—This consists in a combination of baths with exercises at Nauheim. The water has a temperature of from 82°-95° F., and is very richly charged with CO_2. The good effects of the bath are claimed by Schott to come from a cutaneous excitation, induced by the mineral and gaseous constituents of the bath, and a stimulation of the sensory nerves. There is no question that the bath, in suitable cases, will alter the position of the apex beat, and that it lessens the area of cardiac dulness; this means that it diminishes the dilatation of the heart. Artificial baths are used, consisting of forty gallons of water, with various strengths of sodium chloride and calcium chloride. The exercises, resistance gymnastics, consist in slow movements executed by the patient and resisted by the operator. The best cases for the Nauheim treatment are those with myocardial weakness from whatever cause. For valvular heart diseases in the stage of broken compensa-

method of Occ. of mistakes.

tion with dropsy, etc., it is not so suitable. The neurotic heart is often much benefited.

III. ENDOCARDITIS

Inflammation of the lining membrane of the heart is usually confined to the valves, so that the term is practically synonymous with valvular endocarditis. It occurs in two forms—*acute*, characterized by the presence of vegetations with loss of continuity or of substance in the valve tissues; *chronic*, a slow sclerotic change, resulting in thickening, puckering, and deformity.

ACUTE ENDOCARDITIS

This occurs in rare instances as a primary, independent affection; but in the great majority of cases it is an accident in various infective processes, so that in reality the disease does not constitute an etiological entity.

For convenience of description we speak of a simple or benign, and a malignant, ulcerative, or infective endocarditis, between which, however, there is no essential anatomical difference, as all gradations can be traced, and they represent but different degrees of intensity of the same process.

Etiology.—SIMPLE ENDOCARDITIS does not constitute a disease of itself, but is invariably found with some other affection. In 330 cases of rheumatic fever at the Johns Hopkins Hospital there were 110 cases of endocarditis. Bouillaud first emphasized the frequency of the association of simple endocarditis with rheumatic fever. Before him, however, the association had been noticed. Possibly it is nothing in the disease itself, but simply an altered state of the fluid media—a reduction perhaps of the lethal influences which they normally exert—permitting the invasion of the blood by certain micro-organisms. Tonsillitis, which in some forms is regarded as a rheumatic affection, may be complicated with endocarditis. Of the specific diseases of childhood it is not uncommon in scarlet fever, while it is rare in measles and chicken-pox. In diphtheria simple endocarditis is rare. In small-pox it is not common. In typhoid fever it occurred six times among 1,500 cases.

In pneumonia both simple and malignant endocarditis ~~are~~ common. In 100 autopsies in this disease made at the Montreal General Hospital there were 5 instances of the former. Among 61 cases of endocarditis studied bacteriologically in Welch's laboratory, pneumococci were found in 21 (Marshall). Of 517 fatal cases of acute endocarditis, 115 were in connection with pneumonia—22.3 per cent. (E. F. Wells). Acute endocarditis is by no means rare in ~~phthisis~~. ~~I~~ found it in 12 cases in 216 post mortems.

In chorea simple warty vegetations are found on the valves in a large majority of all fatal cases, in 62 of 73 cases collected ~~by me~~. There is no disease in which, post mortem, acute endocarditis has been so frequently found. And, lastly, simple endocarditis is met with in diseases associated with loss of flesh and progressive debility, as cancer, ~~and such disorders as~~ gout, ~~diabetes~~, and ~~Bright's disease~~.

A very common form is that which occurs on the sclerotic valves in old heart-disease—the so-called recurring endocarditis.

MALIGNANT OR INFECTIVE ENDOCARDITIS is met with: (*a*) As a primary disease of the lining membrane of the heart or of its valves.

(*b*) As a secondary affection in rheumatic fever, pneumonia, in various specific fevers, in septic processes of all sorts, and most frequently of all as an infection on old sclerotic valves. In a majority of all cases it is a local process in an acute infection. Congenital lesions are very prone to the severer types of endocarditis, particularly affections of the orifice of the pulmonary artery and the margins of the imperfect ventricular septum (C. Robinson).

The existence of a primary endocarditis has been doubted; but there are instances in which persons previously in good health, without any history of affections with which endocarditis is usually associated, have been attacked with symptoms resembling severe typhus or typhoid. In one case which I saw death occurred on the sixth day and no lesions were found other than those of malignant endocarditis.

The simple endocarditis of rheumatic fever or of chorea rarely progresses into the malignant form. In only 24 of 209 cases the symptoms of severe endocarditis arose in the progress of acute rheumatism. Of all acute diseases complicated with severe endocarditis pneumonia probably heads the list. Gonorrhœa is a much more common cause than has been supposed. There have been at least ten instances in my wards.

The affection may complicate erysipelas, septicæmia (from whatever cause), and puerperal fever. Malignant endocarditis is very rare in tuberculosis, typhoid fever, and diphtheria. In dysentery, in small-pox, and in scarlet fever, with which simple endocarditis is not infrequently complicated, the malignant form is extremely rare.

Morbid Anatomy of Simple and Malignant Endocarditis.—Simple endocarditis is characterized by the presence on the valves or on the lining membrane of the chambers of minute vegetations, ranging from 1 to 4 mm. in diameter, with an irregular and fissured surface, giving to them a warty or verrucose appearance. Often these little cauliflower-like excrescences are attached by very narrow pedicles. They are more common on the left side of the heart than the right, and occur on the mitral more often than on the aortic valves. The vegetations are upon the line of closure of the valves—i. e., on the auricular face of the auriculo-ventricular valves, a little distance from the margin, and on the ventricular side of the sigmoid valves, festooned on either half of the valve from the corpora Arantii. It is rare to see any swelling or macroscopic evidence of infiltration of the endocardium in the neighborhood of even the smallest of the granulations, or of redness, indicative of distention of the vessels, even when they occur upon valves already the seat of sclerotic changes, in which capillary vessels extend to the edges. With time the vegetations may increase greatly in size, but in what may be called simple endocarditis the size rarely exceeds that mentioned above. Hirschfelder has shown experimentally that they may form with great rapidity, even in a few hours.

The earliest vegetations consist of elements derived from the blood, and are composed of blood platelets, leucocytes, and fibrin in varying proportions. At a later stage they appear as small outgrowths of connective tissue. The transition of one form into the other can often be followed. The process consists of a proliferation of the endothelial cells and the cells of the subendothelial layer which gradually invade the fresh vegetation, and ultimately entirely replace it. The blood cells and fibrin undergo disintegration and

Children under 4 years of age should not be admitted.

gradually ~~they~~ are removed. The ~~whole~~ process has received the name of "organization." Even when the vegetation has been entirely converted into connective tissue it is often found at autopsy to be capped with a thin layer of fibrin and leucocytes.

Micro-organisms are generally, even if not invariably, found associated with the vegetations. They tend to be entangled in the granular and fibrillated fibrin or in the older ones to cap the apices.

Subsequent Changes.—(*a*) The vegetations may become organized and the valve restored to a normal state (?). (*b*) The process may extend, and a simple may become an ulcerative endocarditis. (*c*) The vegetations may be broken off and carried in the circulation to distant parts. (*d*) The vegetations become organized and disappear, but they initiate a nutritive change in the valve tissue which ultimately leads to sclerosis, thickening, and deformity. The danger in any case of simple endocarditis is not immediate, but remote, and consists in this perversion of the normal processes of nutrition which results in sclerosis of the valves.

A gradual transition from the simple to a more severe affection, to which the name malignant or ulcerative endocarditis has been given, may be traced. Practically in every case of ulcerative endocarditis vegetations are present. In this form the loss of substance in the valve is more pronounced, the deposition—thrombus formation—from the blood is more extensive, and the micro-organisms are present in greater number and often show increased virulence. Ulcerative endocarditis is often found in connection with heart valves already the seat of chronic proliferative and sclerotic changes.

In this form there is much loss of substance, which may be superficial and limited to the endocardium, or, what is more common, it involves deeper structures, and not very infrequently leads to perforation of a valve, the septum, or even of the heart itself.

Upon microscopic examination the affected valve shows necrosis, with more or less loss of substance; the tissue is devoid of preserved nuclei and presents a coagulated appearance. Upon it a mixture of blood platelets, fibrin—granular or fibrillated—and leucocytes enclosing masses of micro-organisms are met with. The subjacent tissue often shows sclerotic thickening and always infiltration with exuded cells.

Parts Affected.—The following figures, taken from ~~my~~ Goulstonian lectures, give an approximate estimate of the frequency with which in 209 cases different parts of the heart were affected in malignant endocarditis: Aortic and mitral valves together, in 41; aortic valves alone, in 53; mitral valves alone, in 77; tricuspid in 19; the pulmonary valves in 15; and the heart walls in 33. In 9 instances the right heart alone was involved, in most cases the auriculo-ventricular valves.

Mural endocarditis is seen most often at the upper part of the septum of the left ventricle. Next in order is the endocarditis of the left auricle on the postero-external wall. The vegetations may extend, ~~as in a case in my wards~~, along the intima of the pulmonary artery into the hilum of the lung. A common result of the ulceration is the production of valvular aneurism. In three fourths of the cases the affected valves present old sclerotic changes. The process may extend to the aorta, producing, as in one of my cases, extensive endarteritis with multiple acute aneurisms.

Associated Lesions.—The associated changes are those of the primary disease, those due to embolism, and the changes in the myocardium. In the endocarditis of septic processes there is the local lesion—an acute necrosis, a suppurative wound, or puerperal disease. In many cases the lesions are those of pneumonia, rheumatism, or other febrile processes.

The changes due to embolism constitute the most striking features, but it is remarkable that in some instances, even with endocarditis of a markedly ulcerative character, there may be no trace of embolic processes. The infarcts may be few in number—only one or two, perhaps, in the spleen or kidney—or they may exist in hundreds throughout the various parts of the body. They may present the ordinary appearance of red or white infarcts of a suppurative character. They are most common in the spleen and kidneys, though they may be numerous in the brain, and in many cases are very abundant in the intestines. In right sided endocarditis there may be infarcts in the lungs. In many of the cases there are innumerable miliary abscesses. Acute suppurative meningitis was met with in 5 of 23 of the Montreal cases, and in over 10 per cent. of the 209 cases analyzed in the literature. Acute suppurative parotitis also may occur. And lastly, as Romberg has pointed out, the oft accompanying myocarditis plays an important rôle. The valvular insufficiency in an acute endocarditis is probably not due to the row of little vegetations, but to the associated myocarditis, which interferes with the proper closure of the orifice.

Bacteriology.—No distinction in the micro-organisms found in the two forms of endocarditis can be made. In both the pyogenic cocci—streptococci, staphylococci, pneumococci, and gonococci—are the most frequent bacteria met with. More rarely, especially in the simple vegetative endocarditis, the bacilli of tuberculosis, typhoid fever, and anthrax have been encountered. The colon bacillus has also been found, and Howard has described a case of malignant endocarditis due to an attenuated form of the diphtheria bacillus. Marshall in 61 cases found the pneumococci in 21, streptococci alone or with other bacteria in 26, staphylococcus pyogenes aureus in 12. The meningococcus may cause endocarditis, of which there are at least 5 cases in the literature (Cecil and Soper); only 3 of these were associated with meningitis. Combined infections are not uncommon. In the chronic infective form the *Streptococcus mitior* is the common organism (Libman).

As a rule no organisms are found in the simple endocarditis in many chronic diseases, as carcinoma, tuberculosis, nephritis, etc. They may have been present and died out, or the lesions may be caused by the toxins.

Symptoms.—Neither the clinical course nor the physical signs of simple endocarditis are in any respect characteristic. The great majority of the cases are latent and there is no indication whatever of cardiac mischief. Experience has taught us that endocarditis is frequently found post mortem in persons in whom it was not suspected during life. There are certain features, however, by which its presence is indicated with a degree of probability. The patient, as a rule, does not complain of any pain or cardiac distress. In a case of rheumatic fever, for example, the symptoms to excite suspicion would be increased rapidity of the heart's action, perhaps slight irregularity, and an increase in the fever, without aggravation of the joint trouble. Rows of tiny vegetations on the mitral or on the aortic segments seem a trifling

low grade staphylococcus endocarditis—common.
may be active for a time without symptoms.
may give nervous symptoms

3 types from S. viridans. V. nephritis rare in pneumonic
V. chr. endoc. - fever. endocard.
embolism. sign. etc.
3/4 pts. of cases (from endocard.)
anaemia enlarged spleen
thrombosis of aberrans.

matter to excite fever, and it is difficult in the endocarditis of febrile processes to say definitely in every instance that an increase in the fever depends upon this complication; but a study of the recurring endocarditis—which is of the warty variety, consisting of minute beads on old sclerotic valves—shows that the process may be associated, for days or weeks at a time, with slight fever ranging from 100° to 102½°. Palpitation may be a marked feature and is a symptom upon which certain authors lay great stress.

The *diagnosis* of the condition rests upon physical signs, which are notoriously uncertain. The presence of a murmur at one or other of the cardiac areas in a case of fever is often taken as proof of the existence of endocarditis—a common mistake which has arisen from the fact that the *bruit de souffle* or bellows murmur is common to it and to a number of other conditions. At first there may be only a slight roughening of the first sound, which may gradually increase to a distinct murmur. The apex systolic bruit is probably more often the result of a myocarditis. It may not be present in the endocarditis of such chronic maladies as tuberculosis and carcinoma, since in them the muscle involvement is less common (Krehl). Reduplication and accentuation of the pulmonic second sound are frequently present.

It is difficult to give a satisfactory clinical picture of MALIGNANT ENDOCARDITIS because the modes of onset are so varied and the symptoms so diverse. Arising in the course of some other disease, there may be simply an intensification of the fever or a change in its character. In a majority of the cases there are present certain general features, such as irregular pyrexia, sweating, delirium, and gradual failure of strength.

Embolic processes may give special characters, such as delirium, coma, or paralysis from involvement of the brain or its membranes, pain in the side and local peritonitis from infarction of the spleen, bloody urine from implication of the kidneys, impaired vision from retinal hæmorrhage and suppuration, and even gangrene in various parts from the distribution of the emboli.

Two special types of the disease have been recognized—the septic or pyæmic and the typhoid. In some the cardiac symptoms are most prominent, while in others again the main symptoms may be those of an acute affection of the cerebro-spinal system.

The *septic type* is met with usually in connection with an external wound, the puerperal process, or an acute necrosis or gonorrhœa. There are rigors, sweats, irregular fever, and all of the signs of septic infection. The heart symptoms may be completely masked by the general condition, and attention called to them only on the occurrence of embolism. In many cases the features are those of a severe septicæmia, and the organisms may be isolated from the blood. Optic neuritis is not uncommon, and was present in 15 cases of chronic septic endocarditis examined by Faulkner, and in four of these recurrent retinal hæmorrhages were present.

The *typhoid type* is by far the most common and is characterized by a less irregular temperature, early prostration, delirium, somnolence, and coma, relaxed bowels, sweating, which may be of a most drenching character, petechial and other rashes, and occasionally parotitis. The heart symptoms may be completely overlooked, and in some instances the most careful examination has failed to discover a murmur.

Under the *cardiac group*, as suggested by Bramwell, may be considered

those cases in which patients with chronic valve disease are attacked with marked fever and evidence of recent endocarditis. Many such cases present symptoms of the pyæmic and typhoid character and run a most acute course. In others there may be only slight fever or even after a period of high fever recovery takes place.

In what may be termed the *cerebral group* of cases the clinical picture may simulate a meningitis, either basilar or cerebro-spinal. There may be acute delirium or, as in three of the Montreal cases, the patient may be brought into the hospital unconscious. Heineman reports an instance, with autopsy, in which the clinical picture was that of an acute cerebro-spinal meningitis.

Certain special symptoms may be mentioned. The fever is not always of a remittent type, but may be high and continuous. Petechial rashes are very common and render the similarity very strong to certain cases of typhoid and cerebro-spinal fever. In one case the disease was thought to be hæmorrhagic small-pox. Erythematous rashes are not uncommon. The sweating may be most profuse, even exceeding that which occurs in phthisis and ague. Diarrhœa is not necessarily associated with embolic lesions in the intestines. Jaundice has been observed, and cases are on record which were mistaken for acute yellow atrophy.

The heart symptoms may be entirely latent and are not found unless a careful search be made. Even on examination there may be no murmur present. Instances are recorded by careful observers in which the examination of the heart has been negative. Cases with chronic valve disease usually present no difficulty in diagnosis.

The course of the disease is varied, depending largely upon the nature of the primary trouble. Except in the disease grafted upon chronic valvulitis the course is rarely extended beyond five or six weeks. The most rapidly fatal case on record is described by Eberth, the duration of which was scarcely two days.

Chronic Infective Endocarditis.—This is almost always engrafted on an old, sometimes an unrecognized, valve lesion. At first fever is the only symptom; in a few cases there have been chills at onset or recurring chills may arouse the suspicion of malaria. The patient may keep at work for months with a daily rise of temperature, or perhaps an occasional sweat. The heart features may be overlooked. The murmur of the old valve lesion may show no change, and even with the most extensive disease of the mitral cusps the heart's action may be very little disturbed. For months—six, eight, ten, even thirteen!—fever and progressive weakness may be the only symptoms. These are the cases in which, with recurring chills, the diagnosis of malaria is made. With involvement of the aortic segments the signs of a progressive lesion are more common. Embolic features are not common, occurring only toward the close. Ephemeral cutaneous nodes, red raised painful spots on the skin of hands or feet and lasting a few days, rarely occur except in this form. Post mortem there has been found in my cases a remarkable vegetative endocarditis, involving usually the mitral valves, sometimes with much encrusting of the chordæ tendineæ, and large irregular firm vegetations quite different to those of the ordinary ulcerative form of the disease. In some cases the aortic and tricuspid segments are also involved, and the vegetations may extend on to the walls of the heart.

Sub. + Mod.

Diagnosis.—In many cases the detection of the disease is very difficult; in others, with marked embolic symptoms, it is easy. From simple endocarditis it is readily distinguished, though confusion occasionally occurs in the transitional stage, when a simple is developing into a malignant form. The constitutional symptoms are of a graver type, the fever is higher, rigors are common, and septic and typhoid symptoms occur. Perhaps a majority of the cases not associated with puerperal processes or bone disease are confounded with typhoid fever. A differential diagnosis may even be impossible, particularly when we consider that in typhoid fever infarctions and parotitis may occur. The diarrhœa and abdominal tenderness may also be present, which with the stupor and progressive asthenia make a picture not to be distinguished from this disease. Points which may guide us are: The more abrupt onset in endocarditis, the absence of any regularity of the pyrexia in the early stage of the disease, and the cardiac pain. Oppression and shortness of breath may be early symptoms in malignant endocarditis. Rigors, too, are not uncommon. There is a marked leucocytosis in infective endocarditis. Between pyæmia and malignant endocarditis there are practically no differential features, for the disease really constitutes an *arterial pyæmia* (Wilks). In the acute cases resembling malignant fevers the diagnosis is usually made of typhus, typhoid, cerebro-spinal fever, or even of hæmorrhagic small-pox. The intermittent pyrexia, occurring for weeks or months, has led in some cases to the diagnosis of malaria, but this disease can be ~~positively~~ excluded by the blood examination. Blood cultures may aid greatly in the diagnosis.

The cases usually terminate fatally. The instances of recovery are those more subacute forms, the so-called recurring endocarditis developing on old sclerotic valves in cases of chronic heart disease.

Treatment.—We know no measures by which in rheumatic fever, chorea, or the eruptive fevers the onset of endocarditis can be prevented. As it is probable that many cases arise, particularly in children, in mild forms of these diseases, it is well to guard the patients against taking cold and insist upon rest and quiet, and to bear in mind that of all complications an acute endocarditis, though in its immediate effects harmless, is perhaps the most serious. This statement is enforced by the observations of Sibson that on a system of absolute rest the proportion of cases of rheumatic fever attacked by endocarditis was less than of those who were not so treated. It is doubtful whether in rheumatic fever the salicylates have an influence in reducing the liability to endocarditis. Considering the extremely grave after results of simple endocarditis in children, the question arises whether it is possible to do anything to avert the onset of progressive sclerosis of the affected valve. Caton recommends a systematic plan of treatment: (1) Prolonged rest in bed, three months, to keep the heart quiet; (2) a series of small blisters over the heart; and (3) the iodide of potassium in moderate doses for many months. If there is much vascular excitement aconite may be given and an ice bag placed over the heart. The salicylates are strongly advised by some writers. The treatment of malignant endocarditis is practically that of septicæmia—useless and hopeless in a majority of the cases. Blood cultures should be taken as soon as possible and a vaccine prepared. Horder and others have reported good results. Personally I ~~have not seen a successful case.~~

CHRONIC ENDOCARDITIS

Definition.—A sclerosis of the valves leading to shrinking, thickening, and adhesion of the cusps, often with the deposition of lime salts, with shortening and thickening of the chordæ tendineæ, leading to insufficiency and to narrowing of the orifice. It may be primary, but is oftener secondary to acute endocarditis, particularly the rheumatic form.

Etiology.—It is a mistake to regard every case of sclerotic valve as a sequel to an acute endocarditis. It is long ago since Roy and Adami called attention to the possibility that sclerosis of the valve segments might be a sequel of high pressure. The preliminary endocarditis may be a factor in weakening the valve, the progressive thickening of which may be a direct consequence of the strain. As age advances the valves begin to lose their pliancy, show slight sclerotic changes and foci of atheroma and calcification. Certain poisons appear capable of initiating the change, such as alcohol, lead, syphilis, and gout, though we are at present ignorant of the way in which they act. The poisons of the specific fevers may initiate the change. A very important factor, particularly in the case of the aortic valves, is the strain of prolonged and heavy muscular exertion. In no other way can be explained the occurrence of sclerosis of these valves in young and middle aged men whose occupations necessitate the overuse of the muscles. In the aortic segments it may be only the valvular part of a general arterio-sclerosis.

The *frequency* with which chronic endocarditis is met with may be gathered from the following figures: In the statistics, amounting to from 12,000 to 14,000 autopsies, reported from Dresden, Würzburg, and Prague, the percentage ranged from four to nine. The relative frequency of involvement of the various valves is thus given in the collected statistics of Parrot: The mitral orifice ~~was involved~~ in 621, the aortic in 380, the tricuspid in 46, and the pulmonary in 11. This gives 57 instances in the right to 1,001 in the left heart.

Morbid Anatomy.—Vegetations in the form in which they occur in acute endocarditis are not present. In the early stage, which we have frequent opportunities of seeing, the edge of the valve is a little thickened and perhaps presents a few small nodular prominences, which in some cases may represent the healed vegetations of the acute process. In the aortic valves the tissue about the corpora Arantii is first affected, producing a slight thickening with an increase in the size of the nodules. The substance of the valve may lose its translucency, and the only change noticeable be a grayish opacity and a slight loss of its delicate tenuity. In the auriculo-ventricular valves these early changes are seen just within the margin and here it is not uncommon to find swellings of a grayish red, somewhat infiltrated appearance, almost identical with the similar structures on the intima of the aorta in arterio-sclerosis. Even early there may be seen yellow or opaque white subintimal fatty degenerated areas. As the sclerotic changes increase, the fibrous tissue contracts and produces thickening and deformity of the segment, the edges of which become round, curled, and incapable of that delicate apposition necessary for perfect closure. A sigmoid valve, for instance, may be narrowed one fourth or even one third across its face, the most extreme grade of insufficiency being induced without any special deformity and without any nar-

rowing of the arterial orifice. In the auriculo-ventricular segments a simple process of thickening and curling of the edges of the valves, inducing a failure to close without forming any obstruction to the normal course of the blood-flow, is less common. Still, we meet with instances at the mitral orifice, particularly in children, in which the edges of the valves are curled and thickened, so that there is extreme insufficiency without any material narrowing of the orifice. More frequently, as the disease advances, the chordæ tendineæ become thickened, first at the valvular ends and then along their course. The edges of the valves at their angles are gradually drawn together and there is a narrowing of the orifice, leading in the aorta to more or less stenosis and in the left auriculo-ventricular orifice—the two sites most frequently involved—to constriction. Finally, in the sclerotic and necrotic tissues lime salts are deposited and may even reach the deeper structures of the fibrous rings, so that the entire valve becomes a dense calcareous mass with scarcely a remnant of normal tissue. The chordæ tendineæ may gradually become shortened, greatly thickened, and in extreme cases the papillary muscles are implanted directly upon the sclerotic and deformed valve. The apices of the papillary muscles usually show marked fibroid change.

In all stages of the process the vegetations of simple endocarditis may be present, and the severer, ulcerative forms are very apt to attack these sclerotic valves.

Chronic *mural* endocarditis produces cicatrical like patches of a grayish white appearance which are sometimes seen on the muscular trabeculæ of the ventricle or in the auricles. It often occurs in association with myocarditis.

The endocarditis of the fetus is usually of the sclerotic form and involves the valves of the right more frequently than those of the left side.

IV. CHRONIC VALVULAR DISEASE

GENERAL INTRODUCTION

Effects of Valve Lesions.—The general influence on the work of the heart may be briefly stated as follows: The sclerosis induces insufficiency or stenosis, which may exist separately or in combination. The narrowing retards in a measure the normal outflow and the insufficiency permits the blood current to take an abnormal course. The result in the former case is difficulty in the expulsion of the normal contents of the chamber through the narrow orifice; in the other, the overfilling of a chamber by blood flowing into it from an improper source as, for instance, in mitral insufficiency, when the left auricle receives blood both from the pulmonary veins and from the left ventricle. In both instances the effect is dilatation of a chamber, and to expel the normal amount of blood from a dilated chamber a relatively greater amount of mechanical energy is required, which by various adjustments the muscle is stimulated to do.

The cardiac mechanism is fully prepared to meet ordinary grades of dilatation which constantly occur during sudden exertion. A man, for instance, at the end of a hundred yard race has his right chambers greatly

dilated and his reserve cardiac power worked to its full capacity. The slow progress of the sclerotic changes brings about a gradual, not an abrupt, insufficiency, and the moderate dilatation which follows is at first overcome by the exercise of the ordinary reserve strength of the heart muscle. Gradually a new factor is introduced. The constant increase in the energy put forth by the heart is a stimulus to the muscle fibres to increase in bulk and probably also in number; the heart hypertrophies, and the effect of the valve lesion becomes, as we say, *compensated*. The equilibrium of the circulation is in this way maintained.

The nature of the process with which we have to deal is graphically illustrated in the accompanying diagram, from Martius. The perpendicular lines in the figures represent the power of work of the heart. While the muscle

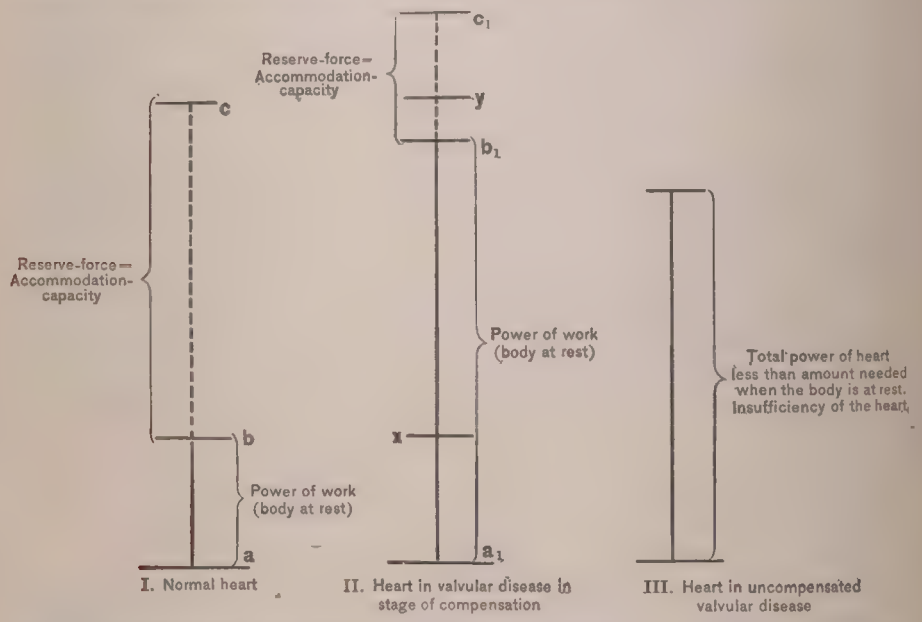

FIG. 6.—DIAGRAMMATIC PRESENTATION SHOWING THE FORCE OF THE HEART FOR WORK UNDER NORMAL CONDITIONS AND IN VALVULAR LESIONS.

in the healthy heart (Diagram I) has at its disposal the maximal force, $a\ c$, it carries on its work under ordinary circumstances (when the body is at rest) with the force $a\ b$ and c is the reserve force by means of which the heart accommodates itself to greater exertion.

If now there be a gross valvular lesion, the force required to do the ordinary work of the heart (at rest) becomes very much increased (Diagram II). But in spite of this enormous call for force, insufficiency of the heart muscle does not necessarily result, for the working force required is still within the limits of the maximal power of the heart, $a_1\ b_1$ being less than $a_1\ c_1$. The muscle accommodates itself to the new conditions by making its reserve force mobile. If nothing further occurred, however, this condition could not be permanently maintained, for there would be left over for emergencies only the small reserve force, $b_1\ y$. Even when at rest the heart would be using

continuously almost its entire maximal force. Any slight exertion requiring more extra force than that represented by the small value $b_1\ y$ (say the effort required on walking or on going upstairs) would bring the heart to the limit of its working power, and palpitation and dyspnœa would appear. Such a condition does not last long. The working power of the heart gradually increases. More and more exertion can be borne without causing dyspnœa, for *the heart hypertrophies.* Finally, a new, more or less permanent condition is attained, in that the hypertrophied heart possesses the maximal force, $a_1\ c_1$. Owing to the increase in volume of the heart muscle, the total force of the heart is greater *absolutely* than that of the normal heart by the amount $y\ c_1$. It is, however, *relatively* less efficient, for its reserve force is much less than that of the healthy heart. Its capacity for accommodating itself to unusual calls upon it is accordingly permanently diminished.

Turning now to the disturbances of compensation, it is to be distinctly borne in mind that any heart, normal or diseased, can become insufficient whenever a call upon it exceeds its maximal working capacity. The liability to such disturbance will depend, above all, upon the accommodation limits of the heart—the less the width of the latter, the easier will it be to go beyond the heart's efficiency. A comparison of Diagrams I and II will immediately make it clear that the heart in valvular disease will much earlier become insufficient than the heart of a healthy individual. If the heart muscle is compelled to do maximal or nearly maximal work for a long time, it becomes exhausted. It is obvious that the heart in valvular disease, on account of its small amount of reserve force, has to do maximal or nearly maximal work far more frequently than does the normal heart. The power of the heart may become decreased to the amount necessary simply to carry on the work of the heart when the body is at rest, or it may cease to be sufficient even for this. The reserve force gained through the compensatory process may be entirely lost (Diagram III). If the loss be only temporary, the exhausted heart muscle quickly recovering, the condition is spoken of as a "disturbance of compensation." The term "loss of compensation" is reserved for the condition in which the disturbance is continuous.

The schema of Martius (Fig. 7) will enable the student to understand the relation of the pathological phenomena to the normal cardiac cycle. The contraction of the ventricle takes an appreciable period of time, seven-hundredths of a second (*a-b*) to overcome the strong arterial pressure which keeps the aortic (and pulmonary) doors tightly shut. This closure-time is the only brief period in the cycle in which both the auriculo-ventricular valves and the semilunar valves are shut, the former as a result of the beginning of the systole, the latter until the intra-ventricular has overcome the aortic pressure. With this closure-time correspond the first sound and the heart beat. In the second period of the ventricular systole the blood is driven into the arteries—the expulsion-time (*b-c*)—and this corresponds with the beginning of the aortic pulse. During this there may be seen at the apex in a forcibly beating heart the "back stroke," as Hope called it. Following the expulsion-time there is a brief period—waiting-time (*c-d*)—before the diastole begins. Clinically the murmur of mitral insufficiency (*A*) coincides, at any rate in its beginning, with the closure-time, the murmur of aortic stenosis with the expulsion-time. The semilunar valves close at the moment when

the ventricles begin to relax (*d*) and with this coincides the second sound. At the same moment the auriculo-ventricular valves open. The murmur of aortic insufficiency (*C*) is heard through the first part of the diastole, sometimes more, while the murmur of mitral stenosis (*D*) corresponds with the latter part of the diastole of the ventricles and with the systole of the auricles.

The *incidence* of valvular lesions may be gathered from the following figures compiled by Gillespie from the records of the Royal Infirmary, Edinburgh: Of 2,368 cases with cardiac lesions, valvular disease occurred in 80.8 per cent.; endocarditis and pericarditis in 5.3; myocardial lesions in 11.9 per cent.; 66.2 per cent. of the cases were in males.

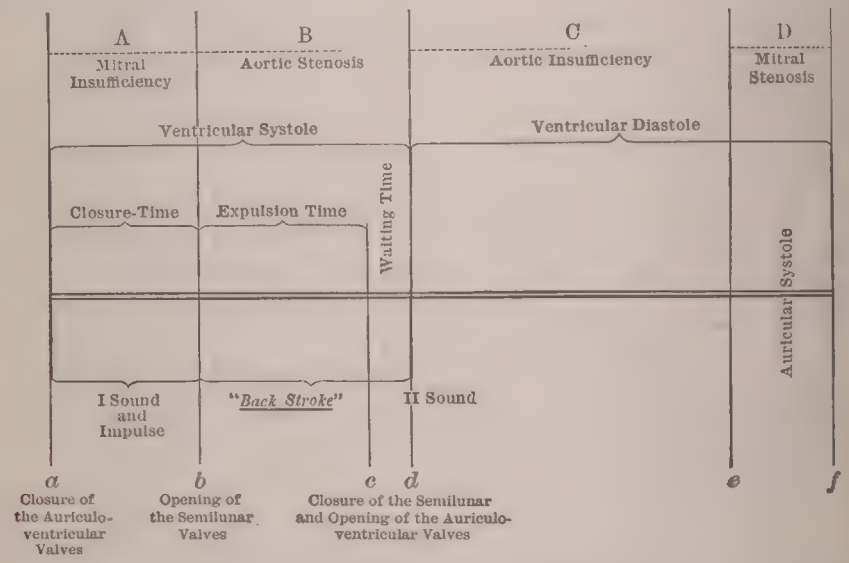

FIG. 7.—SCHEMATIC DIVISION OF THE PHASES OF THE HEART'S ACTION (Martius).

AORTIC INCOMPETENCY

Incompetency of the aortic valves arises either from inability of the valve segments to close an abnormally large orifice or more commonly from disease of the segments themselves. This best-defined and most easily recognized of valvular lesions was first carefully studied by Corrigan, whose name it sometimes bears.

Etiology and Morbid Anatomy.—It is more frequent in males than in females, affecting chiefly able bodied, vigorous men at the middle period of life. The ratio which it bears to other valve diseases has been variously given as from 30 to 50 per cent.

There are five groups of cases: I. Those due to *congenital malformation*, particularly fusion of two of the cusps—most commonly those behind which the coronary arteries are given off. It is probable that an aortic orifice may be competent with this bicuspid state of the valves, but a great danger is the liability of these malformed segments to sclerotic endocarditis. Of 17 cases

Should always be made an etiological diagnosis, other
syphilis. Mem. Spirochaeta pallida in cases of ulcus
ves. is negative.

which I have reported all presented sclerotic changes, and the majority of them had, during life, the clinical features of chronic heart-disease.

II. The *endocarditic group.* Endocarditis may produce an acute insufficiency by ulceration and destruction of the valves; in one case the aortic valves were completely eroded away. The valvulitis of rheumatism and of the fevers, while more rarely aortic, is common enough in children, and the insufficiency is caused by nodular excrescences at the margins or in the valves, which may ultimately become calcified; more often it induces a slow sclerosis of the valves with adhesions, causing also some degree of narrowing.

III. The *arterio-sclerotic group.* By far the most frequent cause of insufficiency is a slow, progressive sclerosis of the segments, resulting in a curling of the edges, which lessens the working surface of the valve. Most frequent in strong, able bodied men, there are three main factors in its production: First, *strain*—not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle. Of circumstances increasing this tension, repeated and excessive use of the muscles is perhaps the most important. So often is this form of heart disease found in persons devoted to athletics that it is sometimes called the "athlete's heart." Secondly, *alcohol,* the action of which is probably direct as a poison to the vessel wall and not, as we have supposed heretofore, in keeping up a high blood pressure. Thirdly, *syphilis,* which is of importance equal to alcohol and strain combined. The Wassermann reaction is present in a very large proportion of all cases of aortic insufficiency in young and middle aged men.

In a small group, usually in young men, syphilis causes a localized arterio-sclerosis at the root of the aorta, involving the valves secondarily or causing dilatation of the aortic ring with relative insufficiency. The endarteritis may be singularly localized, even annular, sometimes patchy. The spirochætes have been found in the lesions.

The condition of the valves is such as has already been described in chronic endocarditis. It may be noted, however, how slight a grade of curling may produce serious incompetency. Associated with the valve disease is, in a majority of cases, a more or less advanced arterio-sclerosis of the arch of the aorta, one serious effect of which may be a narrowing of the orifices of the coronary arteries. The sclerotic changes are often combined with atheroma, either in a fatty or calcareous stage. This may exist at the attached margin of the valves without inducing insufficiency. In other instances insufficiency may result from a calcified spike projecting from the aortic attachment into the body of the valve, and so preventing its proper closure. Some writers (Peter) have laid great stress upon the extension of the endarteritis to the valve, and would separate the instances of this kind from those of simple valvular endocarditis. Anatomically one can usually recognize the arterio-sclerotic variety by the smooth surface, the rounded edges, and the absence of excrescences.

IV. Insufficiency may be induced by *rupture of a segment*—a very rare event in healthy valves, but not uncommon in disease, either from excessive effort during heavy lifting or from the ordinary endarterial strain on a valve eroded and weakened by ulcerative endocarditis.

V. *Relative insufficiency,* due to dilatation of the aortic ring and adjacent

arch, is not very frequent. It occurs in extensive arterial sclerosis of the ascending portion of the arch with great dilatation just above the valves. The valve segments are usually involved with the arterial coats, but the changes in them may be very slight. In aneurism just above the aortic ring relative insufficiency of the valve may be present.

It would appear from the careful measurements of Beneke that the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart until at one and twenty it is about 60 mm. At this it remains until the age of forty, beyond which date there is a gradual increase in the size up to the age of eighty, when it may reach from 68 to 70 mm. There is thus at the ~~very~~ period ~~of life~~ in which sclerosis of the valve is most common a physiological tendency toward the production of ~~a state of~~ relative insufficiency.

The insufficiency may be combined with various grades of narrowing, particularly in the endocarditic group. In a majority of the cases of the arteriosclerotic form there is no stenosis. On the other hand, aortic stenosis almost without exception is associated with some grade, however slight, of regurgitation.

Non-valvular incompetency is met with in two groups of cases, in one of which there is a stretching of the aortic ring in connection with dilatation of the ascending portion of the arch. Whether insufficiency occurs apart from this in cases of dilatation of the left ventricle has been much discussed —a relative incompetency similar to that which occurs at the pulmonary orifice. Cases are reported in which transient diastolic murmurs have occurred ~~in connection~~ with dilatation of the heart, of which Anders ~~has~~ reported and collected corroborative cases. Some years ago J. B. MacCallum, whose untimely death was a great loss to science, described a sphincter-like band of muscle encircling the opening of the left ventricle into the aorta, and in these cases the relaxation of this ring muscle may be associated with insufficiency of the valve.

Effects.—The direct effect of aortic insufficiency is the regurgitation of blood from the artery into the ventricle, causing an overdistention of the cavity and a reduction of the blood column; that is, a relative anæmia in the arterial tree. The amount returning varies with the size of the opening. The double blood flow into the left ventricle causes dilatation of the chamber, and finally hypertrophy, the grade depending upon the lesion. In this way the valve defect is compensated, and, as with each ventricular systole a larger amount of blood is propelled into the arterial system, the regurgitation of a certain amount during diastole does not, for a time at least, seriously impair the nutrition of the peripheral parts. For a time at least there is little or no resistance offered to the blood flow from the auricle—the ventricle accommodates itself readily to the extra amount, and there is no disturbance in the lesser circulation. In acute cases, on the other hand, with rapid destruction of the segments, there may be the most intense dyspnœa and even profuse hæmoptysis. In this lesion dilatation and hypertrophy reach their most extreme limit. The heaviest hearts on record are described in connection with this affection. The so-called bovine heart, *cor bovinum*, may weigh 35 or 40 ounces, or even, as in a case of Dulles's, 48 ounces. The dilatation is usually extreme and is in marked contrast to the condition of the chamber in cases of

pure aortic stenosis. The papillary muscles may be greatly flattened. The mitral valves are usually not seriously affected, though the edges may present slight sclerosis, and there is often relative incompetency, owing to distention of the mitral ring. Dilatation and hypertrophy of the left auricle are common, and secondary enlargement of the right heart occurs in all cases of long standing. In the arterio-sclerotic group there is an ever present possibility of narrowing of the orifices of the coronary arteries or an extension of the sclerosis to their branches, leading to fibroid myocarditis. In the endocarditis cases, particularly those following rheumatism, the intima is perfectly smooth, and the arch with its main branches not dilated. A normal aorta may be found post mortem when during life there have been the most characteristic signs of enlargement of the arch and of dilatation of the innominate and right carotid. The so-called dynamic dilatation of the arch is best seen in these cases. A young girl, whose case had been reported as one of aneurism, had forcible pulsation and a tumor which could be grasped above the sternum —post mortem the innominate artery did not admit the little finger and the arch was not dilated!

Although the coronary arteries, as shown by Martin and Sedgwick, are filled during the ventricular systole, the circulation in them must be embarrassed in aortic incompetency. They must miss the effect of the blood pressure in the sinuses of Valsalva during the elastic recoil of the arteries, which surely aids in keeping the coronary vessels full. The arteries of the body usually present more or less sclerosis consequent upon the strain which they undergo during the forcible ventricular systole.

Symptoms.—The condition is often discovered accidentally in persons who have not presented any features of cardiac disease.

Headache, dizziness, flashes of light, and a feeling of faintness on rising quickly are among the earliest symptoms. Palpitation and cardiac distress on slight exertion are common. Long before any signs of failing compensation pain may become a marked and troublesome feature. It is extremely variable in its manifestations. It may be of a dull, aching character confined to the præcordia. More frequently, however, it is sharp and radiating, and is transmitted up the neck and down the arms, particularly the left. Attacks of true angina pectoris are more frequent in this than in any other valvular disease. Anæmia is also common, much more so than in aortic stenosis or in mitral affections.

As compensation fails more serious symptoms are shortness of breath and œdema of the feet. The attacks of dyspnœa are liable to come on at night, and the patient has to sleep with his head high or even in a chair. Cyanosis is rare. It is most commonly due to complicating valve disease, or it is stated that it may result from bulging of the septum ventriculorum and encroachment upon the right ventricle. Of respiratory symptoms cough is common, due to the congestion of the lungs or œdema. Hæmoptysis is less frequent than in mitral disease. I have reported a case in which it was profuse and believed to be due to tuberculosis of the lungs, inasmuch as the patient was admitted in a state of emaciation and profound exhaustion. General dropsy is not common, but œdema of the feet may occur early and is sometimes due to the anæmia, sometimes to the venous stasis, at times to both. Unless there is coexisting disease of the mitral valve, it is rare in aortic in-

competency for the patient to die with general anasarca. Sudden death is frequent; more so in this than in other valvular diseases. As compensation fails the patient takes to bed and slight irregular fever, associated usually with a recurring endocarditis, is not uncommon toward the close. Embolic symptoms are not infrequent—pain in the splenic region with enlargement of the organ, hæmaturia, and in some cases paralysis. Distressing dreams and disturbed sleep are more common in this than in other forms of valvular disease.

Mental symptoms are often seen with this lesion; toward the close there may be delirium, hallucinations, and morbid impulses. It is important to bear this in mind, for patients occasionally display suicidal tendencies. I have twice had patients throw themselves from a window of the ward.

Physical Signs.—*Inspection* shows a wide and forcible area of cardiac impulse with the apex beat in the sixth or seventh interspace, and perhaps as far out as the anterior axillary line. In young subjects the præcordia may bulge. There may be slight visible pulsation in the second right interspace, or, in some acute cases of insufficiency or ulcerative endocarditis, a couple of inches from the sternal margin. In very slight insufficiency there may be little or no enlargement to be determined clinically. On *palpation* a thrill, diastolic in time, is occasionally felt, but is not common. The impulse is usually strong and heaving, unless in conditions of extreme dilatation, when it is wavy and indefinite. Occasionally two or three interspaces between the nipple line and sternum will be depressed with the systole as the result of atmospheric pressure. *Percussion* shows a greater increase in the area of heart dulness than is found in any other valvular lesion. It extends chiefly downward and to the left.

Auscultation.—A murmur is heard during the diastole of the ventricles at the base of the heart and propagated down the sternum. It may be feeble or inaudible at the aortic cartilage, and is usually heard best at midsternum opposite the third costal cartilage or along the left border of the sternum as low as the ensiform cartilage. It is usually soft, blowing in quality, and is prolonged, or "long drawn," as the phrase is. It is produced by the reflux of blood into the ventricle. In some cases it is loudly transmitted to the axilla at the level of the fourth interspace, not by way of the apex. The second sound may be well heard or it may be replaced by the murmur, or with a dilated and calcified arch the second sound may have a ringing metallic or booming quality, and the diastolic murmur is well heard, or even loudest, over the manubrium.

The first sound may be clear at the base; more commonly there is a soft, short, systolic murmur. In the arterio-sclerotic group the systolic bruit is, as a rule, short and soft, while in the endocarditic group, in which the valve segments are united and often covered with calcified vegetations and excrescences, the systolic murmur is rough and may be accompanied by a thrill.

At the apex, or toward it, the diastolic murmur may be faintly heard propagated from the base. With full compensation the first sound is usually clear at the apex; with dilatation there is a loud systolic murmur of relative mitral insufficiency, which may disappear under observation as the dilatation lessens.

A second murmur at the apex, probably produced at the mitral orifice, is not uncommon. Attention was called to this by the late Austin Flint, and

[illegible] another very unstable and difficult to manage.

the murmur usually goes by his name. It is of a rumbling, echoing character, occurring in the middle or latter part of diastole, usually presystolic in time, and limited to the apex region. It is similar to, though less intense than, the louder presystolic murmurs of mitral stenosis, and is often associated with a palpable thrill. It is probably caused by the impinging of the regurgitant current from the aortic orifice on the large, anterior flap of the mitral valve, so as to cause interference with the entrance of blood at the time of auricular contraction. The condition is thus essentially the same as in a moderate mitral stenosis. This late diastolic echoing or rumbling murmur is present in about half of the cases of uncomplicated aortic insufficiency (Thayer). It is very variable, disappearing and reappearing again without apparent cause. The sharp, valvular first sound and abrupt systolic shock, so common in true mitral stenosis, are rarely present, while the pulse is characteristic of uncomplicated aortic insufficiency.

Arteries.—The examination of the arteries in aortic insufficiency is of great value. Visible pulsation is more commonly seen in the peripheral vessels in this than in any other condition. The carotids may be seen to throb forcibly, the temporals to dilate, and the brachials and radials to expand with each heart-beat. With the ophthalmoscope the retinal arteries are seen to pulsate. Not only is the pulsation evident, but the characteristic jerking quality is apparent. In the throat the throbbing carotids may lead to the diagnosis of aneurism. In many cases the pulsation can be seen in the suprasternal notch, and prominent, forcibly throbbing vessels beneath the right sterno-mastoid muscle. The abdominal aorta may lift the epigastrium with each systole. In severe cases with great hypertrophy, particularly if anæmia is present, the vascular throbbing may be of an extraordinary character,

Fig. 8.—Pulse Tracing in Aortic Insufficiency; an Extra-systole Is Shown.

jarring the whole front of the chest, causing the head to nod, the pulsation may lift the foot when the knees are crossed, and even the tongue may throb rhythmically. To be mentioned with this is the capillary pulse, met very often in the aortic insufficiency, and best seen in the finger nails or by drawing a line upon the forehead, when the margin of hyperæmia on either side alternately blushes and pales. In extreme grades the face or the hand may blush visibly at each systole. It is met with also in profound anæmia, occasionally in neurasthenia, and in health in conditions of great relaxation of the peripheral arteries. Pulsation may also be present in the peripheral veins. On palpation the characteristic ~~water-hammer~~ or Corrigan pulse is felt. In the majority of instances the pulse wave strikes the finger forcibly with a quick jerking impulse, and immediately recedes or collapses. The characters of this are sometimes best appreciated by grasping the arm above the wrist and holding it up. Moreover, the pulse of aortic regurgitation is usually retarded or delayed—i. e., there is an appreciable interval between the

beat of the heart and the pulsation in the radial artery, which varies according to the extent of the incompetence. Occasionally in the carotid artery the second sound is distinctly audible when absent at the aortic cartilage. Indeed, according to Broadbent, it is at the carotid that we must listen for the second aortic sound, for when heard it indicates that the regurgitation is small in amount, and is consequently a very favorable prognostic element. In the larger arteries a systolic thud or shock may be heard and sometimes a double murmur, as pointed out by Duroziez. The systolic pressure is high and the diastolic much decreased. The sphygmographic tracing is very characteristic. The high ascent, the sharp top, the quick drop in which the dicrotic notch and wave are very slightly marked.

The ~~recent~~ studies of Stewart and of W. G. MacCallum have shown that in aortic insufficiency the low position of the dicrotic notch in the descending arm of the pulse wave and the characteristic collapsing character of the pulse are not due, as was formerly supposed, to the regurgitation in the left ventricle, but to the dilatation of the peripheral arteries, which is a sort of protective adaptation under the vaso-motor influences.

Aortic insufficiency may for years be fully *compensated.* Persons do not necessarily suffer any inconvenience, and the condition is often found accidentally. So long as the hypertrophy just equalizes the valvular defect there may be no symptoms and the individual may even take moderately heavy exercise without experiencing sensations of distress, ~~about the heart.~~ The cases which last the longest are those in which the insufficiency follows endocarditis and is not a part of a general arterio-sclerosis. The age ~~of the patient, too,~~ at the time of onset, is a most important consideration, as in youth the lesion is not often from sclerosis, and the coronary arteries are unaffected. Coexistent lesions of the mitral valves tend early to disturb the compensation. Pure aortic insufficiency is consistent with years of average health and with a tolerably active life.

With the onset of myocardial changes, with increasing degeneration of the arteries, particularly with a progressive sclerosis of the arch and involvement of the orifices of the coronary arteries, the compensation becomes disturbed. The insufficiency of the circulation is seen first on the arterial side in occasional faintings, giddiness, or mental irritability and enfeeblement; later there may be mitral regurgitation and embarrassment of the right side of the heart with its usual features. In advanced cases the changes about the aortic ring may be associated with alterations in the cardiac nerves and ganglia, and so introduce an important factor.

AORTIC STENOSIS

Narrowing or stricture of the aortic orifice is not nearly so common as insufficiency. The two conditions, as already stated, may occur together, however, and probably in almost every case of stenosis there is some leakage.

Etiology and Morbid Anatomy.—In the milder grades there is adhesion between the segments, which are so stiffened that during systole they can not be pressed back against the aortic wall. The process of cohesion between the segments may go on without great thickening, and produce a condition in which the orifice is guarded by a comparatively thin membrane, on the aortic

face of which may be seen the primitive raphes separating the sinuses of Valsalva. In some instances this membrane is so thin and presents so few traces of atheromatous or sclerotic changes that the condition looks as if it had originated during fetal life. More commonly the valve segments are thickened and rigid, and have a cartilaginous hardness. In advanced cases they may be represented by stiff, calcified masses obstructing the orifice, through which a circular or slit like passage can be seen. The older the patient the more likely it is that the valves will be rigid and calcified.

We may speak of a relative stenosis of the aortic orifice when with normal valves and ring the aorta immediately beyond is greatly dilated. A stenosis due to involvement of the aortic ring in sclerotic and calcareous changes without lesion of the valves is referred to by some authors. I have never met with an instance of this kind. A subvalvular stenosis, the result of endocarditis in the mitro-sigmoidean sinus, usually occurs as the result of fetal endocarditis. In comparison with aortic insufficiency, stenosis is a rare disease. It is usually met with at a more advanced period of life than insufficiency, and the most typical cases of it are found associated with extensive calcareous changes in the arterial system in old men.

Owing to the impeded blood flow the ventricle has to work against an increased resistance and its walls become hypertrophied, usually at first with little or no dilatation. We see in this condition the most typical instances of what is called concentric hypertrophy, in which, without much, if any, enlargement of the cavity, the walls are greatly thickened, in contradistinction to the so-called eccentric hypertrophy, in which, with the increase in the thickness of the walls, the chamber itself is greatly dilated. The systole is prolonged, even as much as twenty-five per cent. There may be no changes in the other cardiac cavities if compensation is well maintained; but with its failure come dilatation, impeded auricular discharge, pulmonary congestion, and increased work for the right heart. The arterial changes are, as a rule, not so marked as in aortic insufficiency, for the walls have not to withstand the impulse of greatly increased blood-wave with each systole. On the contrary, the amount of blood propelled through the narrow orifice may be smaller than normal, though when compensation is fully established the pulse wave may be of medium volume.

Symptoms.—Physical Signs.—*Inspection* may fail to reveal any area of cardiac impulse. Particularly is this the case in old men with rigid chest walls and large emphysematous lungs. Under these circumstances there may be a high grade of hypertrophy without any visible impulse. Even when the apex beat is visible, it may be, as Traube pointed out, feeble and indefinite. In many cases the apex is seen displaced downward and outward, and the impulse looks strong and forcible.

Palpation reveals in many cases a thrill at the base of the heart of maximum force in the aortic region. With no other condition do we meet with thrills of greater intensity. The apex beat may not be palpable under the conditions above mentioned, or there may be a slow, heaving, forcible impulse.

Percussion never gives the same wide area of dulness as in aortic insufficiency. The extent of it depends largely on the state of the lungs, whether emphysematous or not.

Auscultation.—A rough systolic murmur, of maximum intensity at the

aortic cartilage, and propagated into the great vessels, is the most constant physical sign in aortic stenosis. One of the last lessons learned by the student of physical diagnosis is to recognize that the systolic murmur at the aortic area does not necessarily mean obstruction at the orifice. Roughening of the valves, or of the intima of the aorta, and hæmic states are much more frequent causes. In aortic stenosis the murmur often has a much harsher quality, is louder, and is more frequently musical than in the conditions just mentioned. When compensation fails and the ventricle is dilated and feeble, the murmur may be soft and distant. The second sound is rarely heard at the aortic cartilage, owing to the thickening and stiffness of the valve. A diastolic murmur is not uncommon, but in many cases it can not be heard. Occasionally, as noted by W. H. Dickinson, there is a musical murmur of greatest intensity in the region of the apex, due probably to a slight regurgitation at high pressure through the mitral valves. The pulse in pure aortic stenosis is small, usually of good tension, well sustained, regular, and perhaps slower than normal.

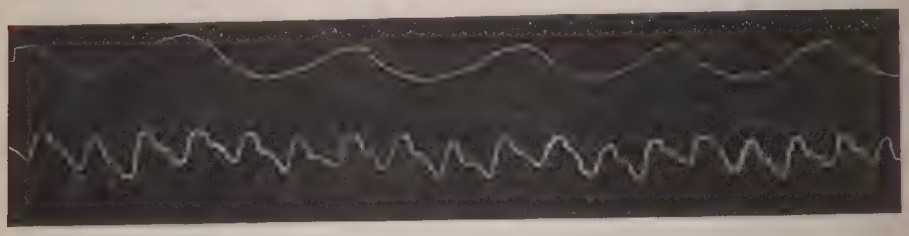

Fig. 9.—Pulse Tracing in Aortic Stenosis.

The condition may be latent for an indefinite period, as long as the hypertrophy is maintained. Early symptoms are those due to defective blood supply to the brain, dizziness, and fainting. Palpitation, pain about the heart, and anginal symptoms are not so marked as in insufficiency. With degeneration of the heart-muscle and dilatation relative insufficiency of the mitral valve is established, and the patient may present all the features of engorgement in the lesser and systemic circulations, with dyspnœa, cough, rusty expectoration, and the signs of anasarca in the lower part of the body. Many of the cases in old people, without presenting any dropsy, have symptoms pointing rather to general arterial disease. Cheyne-Stokes breathing is not uncommon with or without signs of uræmia.

Diagnosis.—With an extremely rough or musical murmur of maximum intensity at the aortic region and signs of hypertrophy of the left ventricle, a thrill, and especially a hard, slow pulse of moderate volume and fairly good tension, which in a sphygmographic tracing gives a curve of slow rise, a broad, well sustained summit and slow decline, a diagnosis of aortic stenosis can be made with some degree of certainty, particularly if the subject is an old man. Mistakes are common, however, and a roughened or calcified valve segment, or, in some instances, a very roughened and prominent calcified plate in the aorta, and hypertrophy associated with renal disease, may produce similar symptoms. Seldom is there difficulty in distinguishing the murmur due to anæmia, since it is rarely so intense and is not associated with thrill or with marked hypertrophy of the left ventricle. In aortic insufficiency a

systolic murmur is usually present, but has neither the intensity nor the musical quality, nor is it accompanied with a thrill. With roughening and dilatation of the ascending aorta the murmur may be very harsh or musical; but the existence of a second sound, accentuated and ringing in quality, is usually sufficient to differentiate this condition.

MITRAL INCOMPETENCY

Etiology.—Insufficiency of the mitral valve ensues: (*a*) From changes in the segments whereby they are contracted and shortened, usually combined with changes in the chordæ tendineæ, or with more or less narrowing of the orifice. (*b*) As a result of changes in the muscular walls of the ventricle, either dilatation, so that the valve segments fail to close an enlarged orifice, or changes in the muscular substance, so that the segments are imperfectly coapted during the systole—muscular incompetency. The common lesions producing insufficiency result from endocarditis, which causes a gradual thickening at the edges of the valves, contraction of the chordæ tendineæ, and union of the edges of the segments, so that in a majority of the instances there is not only insufficiency, but some grade of narrowing as well. Except in children, we rarely see the mitral leaflets curled and puckered without narrowing of the orifice. Calcareous plates at the base of the valve may prevent perfect closure of one of the segments. In long-standing cases the entire mitral structures are converted into a firm calcareous ring. From this valvular insufficiency the other condition of muscular incompetency must be carefully distinguished. It is met with in all conditions of extreme dilatation of the left ventricle, and also in weakening of the muscles in prolonged fevers and in anæmia.

Morbid Anatomy.—The effects of incompetency of the mitral segment upon the heart and circulation are as follows: (*a*) The imperfect closure allows a certain amount of blood to regurgitate from the ventricle into the auricle, so that at the end of auricular diastole this chamber contains not only the blood which it has received from the lungs, but also that ~~which has~~ regurgitated from the left ventricle. This necessitates dilatation, and, as increased work is thrown upon it in expelling the augmented contents, hypertrophy as well.

(*b*) With each systole of the left auricle a larger volume of blood is forced into the left ventricle, which ~~also~~ dilates and subsequently ~~becomes~~ hypertrophied.

(*c*) During the diastole of the left auricle, as blood is regurgitated into it from the left ventricle, the pulmonary veins are less readily emptied. In consequence the right ventricle expels its contents less freely, and in turn becomes dilated and hypertrophied.

(*d*) Finally, the right auricle also is involved, its chamber is enlarged, and its walls are increased in thickness.

(*e*) The effect upon the pulmonary vessels is to produce dilatation both of the arteries and veins—often in long-standing cases, atheromatous changes; the capillaries are distended, and ultimately the condition of brown induration is produced. Perfect compensation may be effected, chiefly through the hypertrophy of both ventricles, and the effect upon the peripheral circulation may

not be manifested for years, as a normal volume of blood is discharged from the left heart at each systole. The time comes, however, when, owing either to increase in the grade of the incompetency or to failure of the compensation, the left ventricle is unable to send out its normal volume into the aorta. Then there are overfilling of the left auricle, engorgement in the lesser circulation, embarrassed action of the right heart, and congestion in the systemic veins. For years this somewhat congested condition may be limited to the lesser circulation, but finally the right auricle becomes dilated, the tricuspid valves incompetent, and the systemic veins are engorged. This gradually leads to the condition of cyanotic induration in the viscera and, when extreme, to dropsical effusion.

Muscular incompetency, due to impaired nutrition of the mitral and papillary muscles, is rarely followed by such perfect compensation. There may be in acute destruction of the aortic segments an acute dilatation of the left ventricle with relative incompetency of the mitral segments, great dilatation of the left auricle, and intense engorgement of the lungs, under which circumstances profuse hæmorrhage may result. In these cases there is little chance for the establishment of compensation. In cases of hypertrophy and dilatation of the heart, without valvular lesions, but associated with heavy work and alcohol, the insufficiency of the mitral valve may be extreme and lead to great pulmonary congestion, engorgement of the systemic veins, and a condition of cardiac dropsy, which can not be distinguished by any feature from that of mitral incompetency due to lesion of the valve itself. In chronic ~~Bright's disease~~ the hypertrophy of the left ventricle may gradually fail, leading, in the later stages, to relative insufficiency of the mitral valve, and the production of ~~a condition of~~ pulmonary and systemic congestion, similar to that induced by the most extreme grade of lesion of the valve itself. Adherent pericardium, especially in children, may lead to like results.

Symptoms.—During the development of the lesion, unless the incompetency comes on acutely in consequence of rupture of the valve segment or of ulceration, the compensatory changes go hand in hand with the defect, and there are no subjective symptoms. So, also, in the stage of perfect compensation, there may be the most extreme grade of mitral insufficiency with enormous hypertrophy of the heart, yet the patient may not be aware of the existence of heart trouble, and may suffer no inconvenience except perhaps a little shortness of breath on exertion or on going upstairs. It is only when from any cause the compensation has not been perfectly effected, or, having been so, is broken abruptly or gradually, that the patients begin to be troubled. The symptoms may be divided into two groups:

(*a*) The minor manifestations while compensation is still good. Patients with extreme incompetency often have a congested appearance of the face, the lips and ears have a bluish tint, and the venules on the cheeks may be enlarged—signs in many cases very suggestive. In long standing cases, particularly in children, the fingers may be clubbed, and there is shortness of breath on exertion. This is one of the most constant features in mitral insufficiency and may exist for years, even when the compensation is perfect. Owing to the somewhat congested condition of the lungs these patients have a tendency to attacks of bronchitis or hæmoptysis. There may also be palpitation of the heart. As a rule, however, in well balanced lesions in adults,

nephilés

this period of full compensation or latent stage is not associated with symptoms which call the attention to an affection of the heart, and with care the patient may reach old age in comparative comfort without being compelled to curtail seriously his pleasures or his work.

(*b*) Sooner or later comes a period of disturbed or broken compensation, in which the most intense symptoms are those of venous engorgement. There are palpitation, weak, irregular action of the heart, and signs of dilatation. Dyspnœa is an especial feature, and there may be cough. A distressing symptom is the cardiac "sleep-start," in which, just as the patient falls asleep, he wakes gasping and feeling as if the heart were stopping. There is usually a slight cyanosis, and even a jaundiced tint to the skin. The most marked symptoms, however, are those of venous stasis. The overfilling of the pulmonary vessels accounts in part for the dyspnœa. There is cough, often with bloody or watery expectoration, and the alveolar epithelium containing brown pigment-grains is abundant. Dropsical effusion usually sets in, beginning in the feet and extending to the body and the serous sacs. Right sided hydrothorax may recur and require repeated tapping. The urine is usually scanty and albuminous, and contains tube casts and sometimes blood corpuscles. With judicious treatment the compensation may be restored and all the serious symptoms may pass away. Patients usually have recurring attacks of this kind, and die of a general dropsy; or there is progressive dilatation of the heart, ~~and death from asystole~~. Sudden death in these cases is rare. Some cases of mitral disease—stenosis and insufficiency—reach what may be called the hepatic stage, when all the symptoms are due to the secondary changes in the liver.

Physical Signs.—*Inspection.*—In children the prærcordia may bulge and there may be a large area of visible pulsation. The apex beat is to the left of the nipple, in some cases in the sixth interspace, in the anterior axillary line. A localized right ventricle impulse may sometimes be seen below the right costal border in the parasternal line. There may be a wavy impulse in the cervical veins, which are often full, particularly when the patient is recumbent.

Palpation.—A thrill is rare; when present it is felt at the apex, often in a limited area. The force of the impulse may depend largely upon the stage in which the case is examined. In full compensation it is forcible and heaving; when the compensation is disturbed, usually wavy and feeble.

Percussion.—The dulness is increased, particularly in a lateral direction. There is no disease of the valves which produces, in long standing cases, a more extensive transverse area of heart dulness. It does not extend so much upward along the left margin of the sternum as beyond the right margin and to the left of the nipple line.

Auscultation.—At the apex there is a systolic murmur which wholly or partly obliterates the first sound. It is loudest here, and has a blowing, sometimes musical character, particularly toward the latter part. The murmur is transmitted to the axilla and may be heard at the back, in some instances over the entire chest. There are cases in which, as pointed out by Naunyn, the murmur is heard best along the left border of the sternum. Usually in diastole at the apex the loudly transmitted second sound may be heard. Occasionally there is also a soft, sometimes a rough or rumbling presystolic mur-

mur. As a rule, in cases of extreme mitral insufficiency from valvular lesion with great hypertrophy of both ventricles, there is heard only a loud blowing murmur during systole. A murmur of mitral insufficiency may vary a great deal according to the position of the patient. It may be present in the recumbent and absent in the erect posture. In cases of dilatation, particularly when dropsy is present, there may be heard at the ensiform cartilage and in the lower sternal region a soft systolic murmur due to tricuspid regurgitation. An important sign on auscultation is the accentuated pulmonary second sound. This is heard to the left of the sternum in the second interspace, or over the third left costal cartilage.

The pulse, in mitral insufficiency, during the period of full compensation, may be full and regular, often of low tension. Usually with the first onset of the symptoms the pulse becomes irregular, a feature which then dominates the case throughout. There may be no two beats of equal force or volume. Often after the disappearance of the symptoms of failure of compensation the irregularity of the pulse persists.

The three important physical signs then of mitral regurgitation are: (*a*) Systolic murmur of maximum intensity at the apex, which is propagated to the axilla and heard at the angle of the scapula; (*b*) accentuation of the pulmonary second sound; (*c*) evidence of enlargement of the heart, particularly the increase in the transverse diameter, due to hypertrophy of both right and left ventricles.

Diagnosis.—There is rarely any difficulty in the diagnosis of mitral insufficiency. The physical signs just referred to are quite characteristic and distinctive. Two points are to be borne in mind. First, a murmur, systolic in character, and of maximum intensity at the apex, and propagated even to the axilla, does not necessarily indicate incompetency of the mitral valve. There is heard in this region a large group of what are termed accidental murmurs, the precise nature of which is still doubtful. They are probably formed, however, in the ventricle, and are not associated with hypertrophy, or accentuation of the pulmonary second sound.

Second, it is not always possible to say whether the insufficiency is due to lesion of the valve segment or to dilatation of the mitral ring and relative incompetency. Here neither the character of the murmur, the propagation, the accentuation of the pulmonary second sound, nor the hypertrophy assists in the differentiation. The history is sometimes of greater value in this matter than the physical examination. The cases most likely to lead to error are those of the so-called idiopathic dilatation and hypertrophy of the heart (in which the systolic murmur may be of the greatest intensity), and the instances of arterio-sclerosis with dilated heart. Balfour and others, however, maintain that organic disease of the mitral leaflets sufficient to produce incompetency is always accompanied with a certain degree of narrowing of the orifice, so that the only unequivocal proof of the actual disease of the mitral valve is the presence of a presystolic murmur.

MITRAL STENOSIS

Etiology.—There are two groups of cases, one following an acute endocarditis, the other the result of a slow sclerosis of the valves without any

This is often due to

history of rheumatic fever or other infection. It is very much more common in women than in men—in 63 of 80 cases noted by Duckworth, while in 4,791 autopsies at Guy's Hospital during ten years there were 196 cases, of which 107 were females and 89 males (Samways). This is not easy to explain, but there are at least two factors to be considered. Rheumatism prevails more in girls than in boys, and, as is well known, endocarditis of the mitral valve is more common in rheumatism. Chorea, also, as suggested by Barlow, has an important influence, occurring more frequently in girls and being often associated with endocarditis. Anæmia and chlorosis, which are prevalent in girls, have been regarded as possible factors. In a surprising number of cases of what the French call *pure* mitral stenosis no recognizable etiological factor can be discovered. This has been regarded by some writers as favoring the view that they may be of congenital origin, but congenital affections of the mitral valve are notoriously rare. Whooping-cough, with its terrible strain on the heart-valves, may be accountable for certain cases. While met with at all ages, stenosis is certainly most frequent in young adult women.

Morbid Anatomy.—The valve segments and chordæ may be fused together, the result of repeated attacks of endocarditis. The condition varies a good deal, according to the amount of atheromatous change. In many cases the curtains are so welded together and the whole valvular region so thickened that the orifice is reduced to a mere chink—Corrigan's button-hole contraction. In non-endocarditic cases the curtains are not much thickened, but narrowing has resulted from gradual adhesion at the edges, and thickening of the chordæ tendineæ, so that from the auricle it looks cone like—the so-called funnel shaped variety of stenosis. The instances in which the valve segments are very slightly deformed, but in which the orifice is considerably narrowed, are regarded by some as possibly of congenital origin. Occasionally the curtains are in great part free from disease, but the narrowing results from large calcareous masses, which project into them from the ring. The involvement of the chordæ tendineæ is usually extreme, and the papillary muscles may be inserted directly upon the valve. In moderate grades of constriction the orifice will admit the tip of the index finger; in more extreme forms the tip of the little finger; and occasionally one meets with a specimen in which the orifice seems almost obliterated, admitting only a medium sized probe. The heart is not greatly enlarged, rarely weighing more than 14 or 15 ounces. Occasionally, in an elderly person, it may seem only slightly, if at all, enlarged, and again there are instances in which the weight may reach as much as 20 ounces. The left ventricle is usually small, and may look very small in comparison with the right ventricle, which forms the greater portion of the apex. In cases in which with the narrowing there is very considerable incompetency the left ventricle may be moderately dilated and hypertrophied.

It is not uncommon at the examination to find white thrombi in the appendix of the left auricle. Occasionally a large part of the auricle is occupied by an ante-mortem thrombus. Still more rarely the remarkable ball thrombus is found, in which a globular concretion, varying in size from a walnut to a small egg, lies free in the auricle, two examples of which have come under my observation.

The left auricle discharges its blood with greater difficulty and in consequence dilates, and its walls reach three or four times their normal thickness.

Although the auricle is by structure unfitted to compensate an extreme lesion, the probability is that for some time during the gradual production of stenosis the increasing muscular power of the walls is sufficient to counterbalance the defect. In 36 cases of well-marked stenosis Samways found the auricle hypertrophied in 26, dilatation coexisting in 14. Eventually the tension is increased in the pulmonary circulation, owing to impeded outflow from the veins and this to heightened pressure in the pulmonary artery. Extra work is thus thrown on the right ventricle, which gradually hypertrophies. Relative incompetency of the tricuspid and congestion of the systemic veins at last supervene.

Symptoms.—PHYSICAL SIGNS.—*Inspection.*—In children the lower sternum and the fifth and sixth left costal cartilages are often prominent, owing to hypertrophy of the right ventricle. The apex beat may be ill defined. Usually it is not dislocated far beyond the nipple line, and the chief impulse is over the lower sternum and adjacent costal cartilages. Often in thin chested persons there is pulsation in the third and fourth left interspaces close to the sternum. When compensation fails, the præcordial impulse is much feebler, and in the veins of the neck there may be marked systolic regurgitation or the right jugular near the clavicle may stand out as a prominent tumor. In the later stage there is great enlargement with pulsation of the liver or pulmonary pulsation seen in the intercostal spaces.

Palpation reveals in a majority of the cases a characteristic, well defined fremitus or thrill, which is best felt, as a rule, in the fourth or fifth interspace within the nipple line. It is of a rough, grating quality, often peculiarly limited in area, most marked during expiration, and can be felt to terminate in a sharp, sudden shock, synchronous with the impulse. This most characteristic of physical signs is pathognomonic of narrowing of the mitral orifice, and is perhaps the only instance in which the diagnosis of a valvular lesion can be made by palpation alone. The cardiac impulse is felt most forcibly in the lower sternum and in the fourth and fifth left interspaces. The impulse is felt very high in the third and fourth interspaces, or in rare cases even in the second, and it has been thought that in the latter interspace the impulse is due to pulsation of the auricle. It is always the impulse of the conus arteriosus of the right ventricle; even in the most extreme grades of mitral stenosis there is never such tilting forward of the auricle or its appendix as would enable it to produce an impression on the chest wall.

Percussion gives an increase in the cardiac dulness to the right of the sternum and along the left margin; not usually a great increase beyond the nipple line, except in extreme cases, when the transverse dulness may reach from 5 cm. beyond the right margin of the sternum to 10 cm. beyond the nipple line.

Auscultation.—To the inner side of the apex beat, often in a very limited region, there is heard a rough, vibratory or purring murmur, cumulative or crescendo in character, which terminates abruptly in the first sound. By combining palpation and auscultation the purring murmur is found to be synchronous with the thrill and the loud shock with the first sound. The murmur is auricular systolic, due to the blood passing through the narrow orifice. Some have thought it to be early systolic in time, but the majority of observers hold to the former view with Gairdner. The presystolic murmur may occupy the entire period of the diastole, or the middle or only the latter half,

Deus omnia videt

corresponding to the auricular systole. The difference may sometimes be noted between the first and second portions of the murmur, when it occupies the entire time. Often there is a peculiar rumbling or echoing quality, which in some instances is very limited and may be heard only over a single bell-space of the stethoscope. A rumbling, echoing presystolic murmur at the apex is heard in some cases of aortic insufficiency (Flint murmur), occasionally in adherent pericardium with great dilatation of the heart, and in upward dislocation of the organ.

A systolic murmur may be heard at the apex or along the left sternal border, often of extreme softness and audible only when the breath is held. Sometimes the systolic murmur is loud and distinct and is transmitted to the axilla. The second sound in the second left interspace is loudly accentuated, and often reduplicated. It may be transmitted far to the left and be heard with great clearness beyond the apex. In uncomplicated cases of mitral stenosis there are usually no murmurs audible at the aortic region, at which spot the second sound is less intense than at the pulmonary area. In advanced cases at the lower sternum and to the right a systolic tricuspid murmur is sometimes heard. Other points to be noted are the following: The unusually sharp, clear first sound which follows the presystolic murmur, the cause of which is by no means easy to explain. It can scarcely be a valvular sound produced chiefly at the mitral orifice, since it may be heard with great intensity in cases in which the valves are rigid and calcified. It has been suggested by A. E. Sansom and others that it is a loud "snap" of the tricuspid valves caused by the powerful contraction of the greatly hypertrophied right ventricle. Broadbent thinks it may be due to the abrupt contraction of a partially filled left ventricle. The valvular sound may be audible at a distance, as one sits at the bedside of the patient (Graves). In a patient ~~I saw~~ with C. J. Blake the first sound was audible six feet ~~by measurement~~ from the chest wall.

These physical signs, it is to be borne in mind, are characteristic only of the stage in which compensation is maintained. The murmur may be soft, almost inaudible, and only brought out after exertion. Finally there comes a period in which, with the establishment of auricular fibrillation, the presystolic murmur disappears and there is heard in the apex region a sharp first sound, or sometimes a gallop rhythm. The marked systolic shock may be present after the disappearance of the thrill and the characteristic murmur. Under treatment, with gradual recovery of compensation, probably with increasing vigor of contraction of the right ventricle and left auricle, the presystolic murmur reappears. In cases seen at this stage of the disease the nature of the valve lesion may be entirely overlooked. As Mackenzie and Lewis have shown, auricular fibrillation is the rule in the arrhythmia of mitral stenosis.

Stenosis of the mitral valve may for years be efficiently compensated by the hypertrophy of the right ventricle. Many persons with the characteristic physical signs of this lesion present no symptoms. They may for years perhaps be short of breath on going upstairs, but are able to pass through the ordinary duties of life without discomfort. The pulse is smaller in volume than normal, and very often irregular. A special danger of this stage is the recurring endocarditis. Vegetations may be whipped off into the circulation

and, blocking a cerebral vessel, may cause hemiplegia or aphasia, or both. This, unfortunately, is not an uncommon sequence in women. Patients with mitral stenosis may survive this accident for an indefinite period. A woman, above seventy years of age, died in one of my wards at the Philadelphia Hospital, who had been in the almshouse, hemiplegic, for more than thirty years. The heart presented an extreme grade of mitral stenosis which had probably existed at the time of the hemiplegic attack.

Pressure of the enlarged auricle on the left recurrent laryngeal nerve, causing paralysis of the vocal cord on the corresponding side, has been described by Ortner and by Herrick. It is a point to be borne in mind, as the diagnosis of aneurism of the arch of the aorta may be made. On the other hand, Fetterolf and Norris conclude that it is not the pressure of the left auricle directly, but the squeezing of the nerve between the pulmonary artery and the aortic arch, and that the paralysis is due to the neuritis so excited.

Failure of compensation brings in its train the group of symptoms which have been discussed under cardiac insufficiency. Briefly enumerated, they are: Rapid and irregular action of the heart, shortness of breath, cough, signs of pulmonary engorgement, and very frequently hæmoptysis. Attacks of this kind may recur for years. Bronchitis or a febrile attack may cause shortness of breath or slight blueness. Inflammatory affections of the lungs or pleura seriously disturb the right heart, and these patients stand pneumonia very badly. Many, perhaps a majority of, cases of mitral stenosis do not have dropsy. The liver may be greatly enlarged, and in the late stages ascites is not uncommon, particularly in children. General anasarca is most frequently met with in those cases in which there is secondary narrowing of the tricuspid orifice (Broadbent).

TRICUSPID VALVE DISEASE

Tricuspid Regurgitation.—Occasionally this results from acute or chronic endocarditis with puckering; more commonly the condition is one of relative insufficiency, and is secondary to lesions of the valves on the left side, particularly of the mitral. It is met with also in all conditions of the lungs which cause obstruction to the circulation, such as cirrhosis and emphysema, particularly in combination with chronc bronchitis. The symptoms are those of obstruction in the lesser circulation with venous congestion in the systemic veins, such as has already been described in connection with mitral insufficiency. The signs of this condition are:

tri-regurg.

(*a*) Systolic regurgitation of the blood into the right auricle and the transmission of the pulse wave into the veins of the neck. If the regurgitation is slight or the contraction of the ventricle is feeble there may be no venous throbbing, but in other cases there is marked systolic pulsation in the cervical veins. That in the right jugular is more forcible than that in the left. It may be seen both in the internal and the external vein, particularly in the latter. Marked pulsation in these veins occurs only when the valves guarding them become incompetent. Slight oscillations are by no means uncommon, even when the valves are intact. The distention is sometimes enormous, particularly in the act of coughing, when the right jugular at the root of the neck may stand out, forming an extraordinarily prominent ovoid mass. Occasionally the regurgitant pulse wave may be widely transmitted and be

Othathemia.

Enlargement
Hypertr. of vent. Displacement to left: dragging on aortic lig. and
and – + dilatation of pulmonary conus

Pulmonary artery by its own enlargement or enlargement of l. auricle
is pressed against nerve + aortic arch.

Nerve caught + compressed between pericardial [illegible]

seen in the subclavian and axillary veins, and even in the subcutaneous veins over the shoulder, or in the superficial mammary veins.

Regurgitant pulsation through the tricuspid orifice may be transmitted to the inferior cava, and so to the hepatic veins, causing a systolic distention of the liver. This is best appreciated by bimanual palpation, placing one hand over the fifth and sixth costal cartilages and the other in the lateral region of the liver in the mid-axillary line. The rhythmical expansile pulsation may be readily distinguished, as a rule, from the systolic depression of the liver due to communicated pulsation from the left ventricle.

(*b*) The second important sign of tricuspid regurgitation is the occurrence of a systolic murmur of maximum intensity in the lower sternum. It is usually a soft, low murmur, often to be distinguished from a coexisting mitral murmur by differences in quality and pitch, and may be heard to the right as far as the axilla. Sometimes it is very limited in its distribution.

Together these two signs positively indicate tricuspid regurgitation. In addition, the percussion usually shows increase in the area of dulness to the right of the sternum, and the impulse in the lower sternal region is forcible. In the great majority of cases the symptoms are those of the associated lesions. In cirrhosis of the lung and in chronic emphysema the failure of compensation of the right ventricle with insufficiency of the tricuspid not infrequently leads either to acute asystole or to gradual failure with cardiac dropsy.

Tricuspid Stenosis.—The condition is rare both clinically and anatomically, and it is not often recognized during life. Of 26,000 medical admissions in the Johns Hopkins Hospital there were only 8 with either clinical or post mortem diagnosis of this condition; and in a total of 3,500 autopsies, only 5 cases were found, all in females. Of a total of 195 collected cases, there were 141 females, 38 males, 16 sex unknown. In a majority of the cases—104—the mitral and tricuspid were affected together, in 14 the tricuspid alone, in 64 the tricuspid and aortic. A definite history of rheumatism was present in only 66 cases (Futcher).

The diagnosis is not often made; extreme cyanosis and dyspnœa are common, and toward the end the ordinary signs of cardiac failure. Among the important physical signs are presystolic pulsation in the jugular veins and in the enlarged liver. A presystolic thrill may be felt at the tricuspid area with a marked systolic shock. The cardiac dulness is greatly increased to the right, a rumbling presystolic murmur may be present over the lower sternum with an extension to the right border. This, with a very snappy first sound, great increase of dulness to the right, and chronic breathlessness with cyanosis, are the important features.

PULMONARY VALVE DISEASE

Murmurs in the region of the pulmonary valves are extremely common; lesions of the valves are exceedingly rare. Balfour has well called the pulmonic area the region of auscultatory romance. A systolic murmur is heard here under many conditions—(1) very often in health, in thin chested persons, particularly in children, during expiration and in the recumbent posture; (2) when the heart is acting rapidly, as in fever and after exertion; (3) it is a favorite situation of the cardio-respiratory murmur; (4) in anæmic

states; and (5), as mentioned previously, the systolic murmur of mitral insufficiency may be transmitted along the left sternal margin. Actual lesions of the valves of the pulmonary artery are rare.

Stenosis is almost invariably a congenital anomaly, and constitutes one of the most important of the congenital cardiac affections. The valve segments are usually united, leaving a small, narrow orifice. In adults cases occasionally occur. The congenital lesion is commonly associated with patency of the ductus Botalli and imperfection of the ventricular septum. There may also be tricuspid stenosis. Acute endocarditis not infrequently attacks the sclerotic valves.

The physical signs are extremely uncertain. There may be a systolic murmur with a thrill heard best to the left of the sternum in the second intercostal space. This murmur may be very like a murmur of aortic stenosis, but is not transmitted into the vessels. Naturally the pulmonary second sound is weak or obliterated, or may be replaced by a diastolic murmur. Usually there is hypertrophy of the right heart.

Pulmonary Insufficiency.—This rare affection is occasionally due to congenital malformation, particularly fusion of two of the segments. It is sometimes present, as Bramwell has shown, in cases of malignant endocarditis. Barie has collected 58 cases.

The physical signs are those of regurgitation into the right ventricle, but, as a rule, it is difficult to differentiate the murmur from that of aortic insufficiency, though the maximum intensity may be in the pulmonary area. The absence of the vascular features of aortic insufficiency is the most suggestive feature. Both Gibson and Graham Steell have called attention to the possibility of leakage through these valves in cases of great increase of pressure in the pulmonary artery, and to a soft diastolic murmur heard under these circumstances, which Steell calls "the murmur of high pressure in the pulmonary artery."

COMBINED VALVULAR LESIONS

Valvular lesions are seldom single or pure; combined lesions are more common. This is particularly the case in congenital disease. In young children mitral and aortic lesions, the result of rheumatic fever, are common. Pure mitral insufficiency and pure mitral stenosis may exist for years, but in time the tricuspid becomes involved, at first in sclerosis and later narrowing of the orifice. Aortic valve lesions are more commonly uncombined than mitral lesions. The added lesion may be hurtful or helpful. The stenosis which so often accompanies the endocarditic variety may lessen the regurgitation in aortic insufficiency; and a progressive narrowing of the mitral orifice may be beneficial in mitral regurgitation.

Prognosis in Valvular Disease.—The question is entirely one of efficient compensation. So long as this is maintained the patient may suffer no inconvenience, and even with the most serious forms of valve lesion the function of the heart may be little, if at all, disturbed.

Practitioners who are not adepts in auscultation and feel unable to estimate the value of the various heart murmurs should remember that the best judgment of the conditions may be gathered from inspection and palpation.

With an apex beat in the normal situation and regular in rhythm the auscultatory phenomena may be practically disregarded.

A murmur *per se* is of little or no moment in determining the prognosis in any given case. There is a large group of patients who present no other symptoms than a systolic murmur heard over the body of the heart, or over the apex, in whom the left ventricle is not hypertrophied, the heart rhythm is normal, and who ~~may~~ (not have) had rheumatism. Indeed, the condition is accidentally discovered, often during examination for life insurance. Among the conditions influencing prognosis are:

(*a*) AGE.—Children under ten are bad subjects. Compensation is well effected, and they are free from many of the influences which disturb compensation in adults. The coronary arteries are healthy, and nutrition of the heart muscle can be readily maintained. Yet, in spite of this, the outlook in cardiac lesions developing in very young children is usually bad. One reason is that the valve lesion itself is apt to be rapidly progressive, and the limit of cardiac reserve force is in such cases early reached. There seems to be proportionately a greater degree of hypertrophy and dilatation. Among other causes of the risks of this period are to be mentioned insufficient food in the poorer classes, the recurrence of rheumatic attacks, and the existence of pericardial adhesions. The outlook in a child who can be carefully supervised and prevented from damaging himself by overexertion is naturally better than in one who is constantly overtasking his muscles. The valvular lesions which occur at, or subsequent to, the period of puberty are more likely to be permanently and efficiently compensated. Sudden death from heart disease is very rare in children.

(*b*) SEX.—Women bear valve lesions, as a rule, better than men, owing partly to the fact that they live quieter lives, partly to the less common involvement of the coronary arteries, and to the greater frequency of mitral lesions. Pregnancy and parturition are disturbing factors, but are, ~~I think~~, less serious than some writers would have us believe.

(*c*) VALVE AFFECTED.—The relative prognosis of the different valve lesions is very difficult to estimate. Each case must, therefore, be judged on its own merits. Aortic insufficiency is unquestionably the most serious; yet for years it may be perfectly compensated. Favorable circumstances in any case are the moderate grade of hypertrophy and dilatation, the absence of all symptoms of cardiac distress, and the absence of extensive arterio-sclerosis and of angina. The prognosis rests in reality with the condition of the coronary arteries. Rheumatic lesions of the valves, inducing insufficiency, are less apt to be associated with endarteritis at the root of the aorta; and in such cases the coronary arteries may escape for years. On the other hand, when the aortic insufficiency is only a part of an extensive arterio-sclerosis at the root of the aorta, the coronary arteries are almost invariably involved, and the outlook in such cases is much more serious. Sudden death is not uncommon, either from acute dilatation during some exertion, or, more frequently, from blocking of one of the branches of the coronary arteries. The liability of this form to be associated with angina pectoris also adds to its severity. Aortic stenosis is a comparatively rare ~~lesion, most commonly met with~~ in middle aged or elderly men, and is, as a rule, well compensated. In Broadbent's series of cases, in which autopsy showed definite aortic narrowing,

forty years was the average age at death, and the oldest was but fifty-three.

In mitral lesions the outlook on the whole is much more favorable than in aortic insufficiency. Mitral insufficiency, when well compensated, carries with it a better prognosis than mitral stenosis. Except aortic stenosis, it is the only lesion commonly met with in patients over three-score years. It must be borne in mind that the cases which last the longest are those in which the valve orifice is more or less narrowed, as well as incompetent. There is, in reality, no valve lesion so poorly compensated and so rapidly fatal as that in which the mitral segments are gradually curled and puckered until they form a narrow strip around a wide mitral ring—a condition specially seen in children. There are many cases of mitral insufficiency in which the defect is thoroughly balanced for thirty or even forty years, without distress or inconvenience. Even with great hypertrophy and the apex beat almost in the mid-axillary line, there may be little or no distress, and the compensation may be most effective. Women may pass safely through repeated pregnancies, though here they are liable to accidents associated with the severe strain. I ~~have~~ had under observation for many years a patient who had her first attack of rheumatism at the age of fifteen, when she already had a well marked mitral murmur. She first came under my observation over thirty years ago, with signs of hypertrophy of the left ventricle and a loud systolic murmur. She ~~has~~ lived a very active life, has been unusually vigorous, has borne eleven children, and ~~has~~ passed through three subsequent attacks of rheumatism. The loud mitral systolic murmur persists, but she is very well, only a little short of breath on exertion.

In mitral stenosis the prognosis is usually regarded as less favorable. ~~My own~~ experience ~~has led~~ me, however, to place this lesion almost on a level, particularly in women, with ~~the~~ mitral insufficiency. It is found very often in persons in perfect health, who have had neither palpitation nor signs of heart-failure, and who have lived laborious lives. ~~The~~ figures ~~given, too, by Broadbent~~ indicate that the ~~date~~ of death in mitral stenosis is comparatively advanced. Of 53 cases ~~abstracted~~ from the post mortem records of St. Mary's Hospital, thirty three was the age for males, and thirty seven or thirty eight for females. These women, ~~too,~~ pass through repeated pregnancies with safety. There are, of course, those too common accidents, the result of cerebral embolism, which are more likely to occur in this than in other forms.

Hard and fast lines can not be drawn in the question of prognosis in valvular disease. Every case must be judged separately, and all the circumstances carefully balanced. There is no question which requires greater experience and more mature judgment, and even the most experienced ~~are sometimes~~ at fault.

The following conditions justify a favorable prognosis: Good general health and good habits; no exceptional liability to rheumatic or catarrhal affections; origin of the valvular lesion independently of degeneration: existence of the valvular lesion without change for over three years; sound ventricles, of moderate frequency, and general regularity of action; sound arteries, with a normal amount of blood and tension in the smaller vessels; and, lastly, freedom from pulmonary, hepatic, and renal congestion.

Treatment of Valvular Lesions.—(*a*) Stage of Compensation.—Medici-

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nal treatment at this period is not necessary and is often hurtful. A very common error is to administer cardiac drugs, such as digitalis, on the discovery of a murmur or of hypertrophy. If the lesion has been found accidentally, it may be best not to tell the patient, but rather an intimate friend. Often it is necessary, however, to be perfectly frank in order that the patient may take certain preventive measures. He should lead a quiet, regulated, orderly life, free from excitement and worry, and the risk of sudden death makes it imperative that the patient suffering from aortic disease should be specially warned against overexertion and hurry. An ordinary wholesome diet in moderate quantities should be taken; tobacco may be allowed in moderation, but stimulants should be interdicted or used in very small amount. Exercise should be regulated entirely by the feelings of the patient. So long as no cardiac distress or palpitation follows, moderate exercise will prove very beneficial. The skin should be kept active by a daily bath. Hot baths should be avoided and the Turkish bath should be interdicted. In the case of full-blooded, somewhat corpulent individuals, an occasional saline purge should be taken. Patients with valvular lesions should not go into very high altitudes. The act of coition has serious risks, particularly in aortic insufficiency. Knowing that the causes which most surely and powerfully disturb the compensation are overexertion, mental worry, and malnutrition, the physician should give suitable instructions in each case. As it is always better to have the coöperation of an intelligent patient, he should, as a rule, be told of the condition, but in this matter the physician must be guided by circumstances, and there are cases in which reticence is the wiser policy.

(*b*) Stage of Broken Compensation.—The break may be immediate and final, as when sudden death results from acute dilatation or from blocking of a branch of the coronary artery, or it may be gradual. Among the first indications are shortness of breath on exertion or attacks of nocturnal dyspnœa. These are often associated with impaired nutrition, particularly with anæmia, and a course of iron or change of air may suffice to relieve the symptoms.

Irregularity of the action of the heart can not always be termed an indication of failing compensation, particularly in instances of mitral disease. It has greater significance in aortic lesions. Serious failure of compensation is indicated by signs of dilatation of the heart, marked cyanosis, the gallop rhythm, or various forms of arrhythmia, with or without the existence of dropsy. Under these circumstances the same measures are to be carried out as are indicated under treatment in cardiac insufficiency.

V. SPECIAL PATHOLOGICAL CONDITIONS

1. ANEURISM OF THE HEART

Aneurism of a valve results from acute endocarditis, which produces softening or erosion and may lead either to perforation of the segment or to gradual dilatation of a limited area under the influence of the blood pressure. The aneurisms are usually spheroidal and project from the ventricular face of a sigmoid valve. They are much less common on the mitral segments. They frequently rupture and produce extensive destruction and incompetency of the valves.

Aneurism of the walls results from the weakening induced by chronic myocarditis, or occasionally it follows acute mural endocarditis, which more commonly, however, leads to perforation. It has followed a stab wound, a gumma of the ventricle, and, according to some authors, pericardial adhesions. The left ventricle near the apex is usually the seat, this being the situation in which fibrous degeneration is most common. Fifty nine of the 60 cases collected by Legg were situated here. In the early stages the anterior wall of the ventricle, near the septum, sometimes even the septum itself, is slightly dilated, the endocardium opaque, and the muscular tissue sclerotic. In a more advanced stage the dilatation is pronounced and layers of thrombi occupy the sac. Ultimately a large rounded tumor may project from the ventricle and ~~may~~ attain a size equal to that of the heart. Occasionally the aneurism is sacculated and communicates with the ventricle through a very small orifice. The sac may be double, as in the cases of Janeway and Sailer. In the museum of Guy's Hospital there is a specimen showing the wall of the ventricle covered with aneurismal bulgings. Rupture occurred in 7 of the 90 cases collected by Legg.

The symptoms ~~produced by aneurism of the heart~~ are indefinite. Occasionally there is marked bulging in the apex region and the tumor may perforate the chest wall. In mitral stenosis the right ventricle may bulge and produce a visible pulsating tumor below the left costal border, which ~~I have known to be~~ mistaken for cardiac aneurism. When the sac is large and produces pressure upon the heart itself, there may be a marked disproportion between the strong cardiac impulse and the feeble pulsation in the peripheral arteries.

2. RUPTURE OF THE HEART

This rare event is usually associated with fatty infiltration or degeneration of the heart-muscle. In some instances acute softening in consequence of embolism of a branch of the coronary artery, suppurative myocarditis, or a gummatous growth has been the cause. Of 100 cases collected by Quain, fatty degeneration was noted in 77. Two thirds of the patients were over sixty years of age. It may occur in infants. Schaps reports a case in an infant of four months associated with an embolic infarct of the left ventricle. Harvey, in his second letter to Riolan (1649), described the case of Sir Robert Darcy, who had distressing pain in the chest and syncopal attacks with suffocation, and finally cachexia and dropsy. Death occurred in one of the paroxysms. The wall of the left ventricle of the heart was ruptured, "having a rent in it of size sufficient to admit any of my fingers, although the wall itself appeared sufficiently thick and strong."

The rent may occur in any of the chambers, but is found most frequently in the left ventricle on the anterior wall, not far from the septum. The accident usually takes place during exertion. There may be no preliminary symptoms, but without any warning the patient may fall and die in a few moments. Sudden death occurred in 71 per cent. of Quain's cases. In other instances there may be ~~in the cardiac region~~ a sense of anguish and suffocation, and life may be prolonged for several hours. In a Montreal case, ~~which I examined,~~ the patient walked up a steep hill after the onset of the symptoms.

and lived for thirteen hours. A case is on record in which the patient lived for eleven days.

3. NEW GROWTHS AND PARASITES

Tubercle and syphilis have already been considered. Primary cancer or sarcoma is extremely rare. Secondary tumors may be single or multiple, and are usually unattended with symptoms, even when the disease is most extensive. In one case I found in the wall of the right ventricle a mass which involved the anterior segment of the tricuspid valve and partly blocked the orifice. The surface was eroded and there were numerous cancerous emboli in the pulmonary artery. In another instance the heart was greatly enlarged, owing to the presence of innumerable masses of colloid cancer the size of cherries. The mediastinal sarcoma may penetrate the heart, though it is remarkable how extensive the disease of the mediastinal glands may be without involvement of the heart or vessels.

Cysts in the heart are rare. They are found in different parts, and are filled either with a brownish or a clear fluid. Blood cysts occasionally occur.

The parasites have been discussed under the appropriate section, but it may be mentioned here that both the *cysticercus cellulosæ* and the echinococcus cysts occur occasionally.

4. WOUNDS AND FOREIGN BODIES

Wounds of the heart may be caused by external injuries, as stabs and bullet wounds, by foreign bodies passing from the gullet or œsophagus, or by puncture for therapeutic purposes.

(*a*) Bullet wounds of the heart are common. Recovery may take place, and bullets have been found encysted in the organ. Stab wounds are still more common. A medical student, while on a spree, passed a pin into his heart. The pericardium was opened, and the head of the pin was found outside of the right ventricle. It was grasped and an attempt made to remove it, but it was withdrawn into the heart and, it is said, caused the patient no further trouble (Moxon). In recent stab wounds the practice now is to expose the heart and attempt to suture the wound. The results have been progressively improving. In a case of stab wound Pagenstecher tied the left coronary artery, which had been divided.

(*b*) Hysterical girls sometimes swallow pins and needles, which, passing through the œsophagus and stomach, are found in various parts of the body. A remarkable case is reported by Allen J. Smith of a girl from whom several dozen needles and pins were removed, chiefly from subcutaneous abscesses. Several years later she developed symptoms of chronic heart disease. At the post mortem needles were found in the tissues of the adherent pericardium, and between thirty and forty were embedded in the thickened pleural membranes of the left side.

(*c*) Puncture of the heart (cardiocentesis) has been recommended as a therapeutic procedure, as in chloroform narcosis, and experimental evidence has been brought forward by B. A. Watson in favor of the operation. He advises abstraction of blood in combination with the puncture, cardiocentesis. The proceeding is not without risk. Hæmorrhage may take place from the

puncture, though it is not often extensive. Sloane has ~~recently~~ urged its use in all cases of asphyxia and in suffocation by drowning and from coal gas. The successful case which he reports illustrates forcibly its stimulating action.

VI. CONGENITAL AFFECTIONS OF THE HEART

These have only a limited clinical interest, as in a large proportion of the cases the anomaly is not compatible with life, and in others nothing can be done to remedy the defect or even to relieve the symptoms.

The congenital affections result from interruption of the normal course of development or from inflammatory processes—endocarditis; sometimes from a combination of both.

General Anomalies.—Of general anomalies of development the following conditions may be mentioned: *Acardia,* absence of the heart, which has been met with in the monstrosity known by the same name; *double heart,* which has occasionally been found in extreme grades of fetal deformity; *dextrocardia,* in which the heart is on the right side, either alone or as part of a general transportation of the viscera; *ectopia cordis,* a condition associated with fission of the chest wall and of the abdomen. The heart may be situated in the cervical, pectoral, or abdominal regions. Except in the abdominal variety, the condition is very rarely compatible with extra-uterine life. Occasionally, as in a case reported by Holt, the child lives for some months, and the heart may be seen and felt beating beneath the skin in the epigastric region. This infant was five months old at the date of examination.

Anomalies of the Cardiac Septa.—The septa of both auricles and ventricles may be defective, in which case the heart consists of but two chambers, the *cor biloculare* or reptilian heart. In the septum of the auricles there is a very common defect, owing to the fact that the membrane closing the foramen ovale has failed at one point to become attached to the ring, and leaves a valvular slit which may be large enough to admit the handle of a scalpel. Neither this nor the small cribriform perforations of the membrane are of any significance.

The foramen ovale may be patent without a trace of membrane closing it. In some instances this exists with other serious defects, such as stenosis of the pulmonary artery, or imperfection of the ventricular septum. In others the patent foramen ovale is the only anomaly, and in many instances it does not appear to have caused any embarrassment, as the condition has been found in persons who have died of various affections. The ventricular septum may be absent, the condition known as trilocular heart. Much more frequently there is a small defect in the upper portion of the septum, either in the situation of the membranous portion known as the "undefended space" or in the region situated just anterior to this. The anomaly is very frequently associated with narrowing of the pulmonary orifice or of the conus arteriosus of the right ventricle.

Apart from the instances in association with narrowing of the orifice of the pulmonary artery, or of the conus, there are cases in which defect of the membranous septum is the only lesion, a condition not incompatible with long and fairly active life. The late Professor Brooks of the Johns Hopkins University knew from early manhood that he had heart trouble, but he ac-

complished an extraordinary amount of work, and lived to be about 60. Imperfect septum was the only lesion. The physical signs are fairly distinctive, with usually some enlargement of the heart, and a murmur first described by Roger in the following terms: "It is a loud murmur, audible over a large area, and, commencing with systole, is prolonged so as to cover the normal tic-tac. It has its maximum, not at the base to the right, as in aortic stenosis, or to the left, as in pulmonary stenosis, but at the upper third of the præcordial region. It is central, like the septum, and from this central point gradually diminishes in intensity in every direction. The murmur does not vary at any time, and it is not conducted into the vessels." In several of my cases there has been a distinct systolic intensification of this loud continuous murmur.

Anomalies and Lesions of the Valves.—Numerical anomalies of the valves are not uncommon. The semilunar segments at the arterial orifices are not infrequently increased or diminished in number. Supernumerary segments are more frequent in the pulmonary artery than in the aorta. Four, or sometimes five, valves have been found. The segments may be of equal size, but, as a rule, the supernumerary valve is small.

Instead of three there may be only two semilunar valves, or, as it is termed, the *bicuspid condition*. In my experience this is more frequent in the aortic valve. Of 21 instances only 2 occurred at the pulmonary orifice. Two of the valves have united, and from the ventricular face show either no trace of division or else a slight depression indicating where the union has occurred. From the aortic side there is usually to be seen some trace of division into two sinuses of Valsalva. There has been a discussion as to the origin of this condition, whether it is really an anomaly or whether it is not due to endocarditis, fetal or post-natal. The combined segment is usually thickened, but the fact that this anomaly is met with in the fetus without a trace of sclerosis or endocarditis shows that it may, in some cases at least, result from a developmental error.

Clinically this is a very important congenital defect, owing to the liability of the combined valve to sclerotic changes. Except two fetal specimens, all of my cases showed thickening and deformity, and in 15 of those which I have reported death resulted directly or indirectly from the lesion.

The little fenestrations at the margins of the sigmoid valves have no significance; they occur in a considerable proportion of all bodies.

Anomalies of the auriculo-ventricular valves are not often met with.

FETAL ENDOCARDITIS may occur either at the arterial or auriculo-ventricular orifices. It is nearly always of the chronic or sclerotic variety. Very rarely, indeed, is it of the warty or verrucose form. There are little nodular bodies, sometimes six or eight in number, on the mitral and tricuspid segments—the nodules of Albini—which represent the remains of fetal structures, and must not be mistaken for endocardial outgrowths. The little rounded, bead like hæmorrhages of a deep purple color, which are very common on the heart valves of children, are also not to be mistaken for the products of endocarditis. In fetal endocarditis the segments are usually thickened at the edges, shrunken, and smooth. In the mitral and tricuspid valves the cusps are found united and the chordæ tendineæ are thickened and shortened. In the semilunar valves all trace of the segments has disappeared, leaving a

stiff membranous diaphragm perforated by an oval or rounded orifice. It is sometimes very difficult to say whether this condition has resulted from fetal endocarditis or whether it is an error in development. In very many instances the processes are combined; an anomalous valve becomes the seat of chronic sclerotic changes, and, according to Rauchfuss, endocarditis is more common on the right side of the heart only because the valves are here more often the seat of developmental errors.

Lesions at the Pulmonary Orifice.—*Stenosis* of this orifice is one of the commonest and most important of congenital heart affections. A slow endocarditis causes gradual union of the segments and narrowing of the orifice to such a degree that it admits only the smallest sized probe. In some of the cases the smooth membranous condition of the combined segments is such that it would appear to be the result of faulty development. In some instances vegetations occur. The condition is compatible with life for many years, and in a considerable proportion of the cases of congenital heart disease above the tenth year this lesion is present. With it there may be defect of the ventricular septum. Pulmonary tuberculosis is a very common cause of death. Obliteration or *atresia* of the pulmonary orifice is a less frequent but more serious condition than stenosis. It is associated with persistence of the ductus arteriosus, together with patency of the foramen ovale or defect of the ventricular septum with hypertrophy of the right heart. *Stenosis of the conus arteriosus* of the right ventricle exists in a considerable proportion of the cases of obstruction at the pulmonary orifice. At the outset a developmental error, it may be combined with sclerotic changes. The ventricular septum is imperfect, the foramen ovale is usually open, and the ductus arteriosus patent. These three lesions at the pulmonary orifice constitute the most important group of all congenital cardiac affections. Of 181 instances of various congenital anomalies collected by Peacock, 119 cases came under this category, and, according to this author, in 86 per cent. of the patients living beyond the twelfth year the lesion is at this orifice.

Congenital lesions of the aortic orifice are not very frequent. Rauchfuss has collected 24 cases of stenosis and atresia; stenosis of the left conus arteriosus may also occur, a condition which is not incompatible with prolonged life. Ten of the 16 cases tabulated by Dilg were over thirty years of age.

Transposition of the large arterial trunks is a not uncommon anomaly. There may be neither hypertrophy, cyanosis, nor heart murmur.

Symptoms of Congenital Heart Disease.—Cyanosis occurs in over 90 per cent. of the cases, and forms so distinctive a feature that the terms "blue disease" and "morbus cæruleus" are practically synonyms for congenital heart-disease. The lividity in a majority of cases appears only within the first week of life, and may be general or confined to the lips, nose, and ears, and to the fingers and toes. In some instances there is in addition a general dusky suffusion, and in the most extreme grades the skin is almost purple. It may vary a good deal and may be intense only on exertion. The external temperature is low. Dyspnœa on exertion and cough are common symptoms. A great increase in the number of the red corpuscles has been noted, ~~by Gibson and by Vaquez.~~ In a case of Gibson's there were above eight millions of red blood corpuscles to the cubic millimetre. The children rarely thrive, and often

display a lethargy of both mind and body. The fingers and toes are clubbed to a degree rarely met with in any other affection. The cause of the cyanosis has been much discussed. Morgagni referred it to the general congestion of the venous system due to obstruction, and this view was supported in a paper, one of the ablest that has been written on the subject, by Moreton Stillé. Morrison's analysis of 75 cases of congenital heart disease shows that closure of the pulmonary orifice with patency of the foramen ovale and the ventricular septum is the condition most frequently associated with cyanosis, and he concludes that the deficient aeration of the blood owing to diminished lung function is the most important factor. Another view, often attributed erroneously to William Hunter, was that the discoloration was due to the admixture in the heart of venous and arterial blood; but lesions may exist which permit of very free mixture without producing cyanosis. The question of the cause of cyanosis ~~really~~ can not be considered as settled. Variot ~~has recently~~ made the suggestion that the cause is not entirely cardiac, but is associated with disturbance throughout the whole circulatory system, and particularly a vaso-motor paresis and malaeration of the red blood corpuscles.

Diagnosis.—In the case of children, cyanosis, with or without enlargement of the heart, and the existence of a murmur, are sufficient, as a rule, to determine the presence of a congenital heart lesion. The cyanosis gives us no clew to the precise nature of the trouble, as it is a symptom common to many lesions and it may be absent in certain conditions. The murmur is usually systolic, ~~in character.~~ It is, ~~however,~~ not always present, and there are instances ~~on record~~ of complicated congenital lesions in which the examination showed normal heart sounds. In two or three instances fetal endocarditis has been diagnosed *in gravida* by the presence of a rough systolic murmur, and the condition ~~has been~~ corroborated subsequent to the birth of the child. Hypertrophy is present in a majority of the cases of congenital defect. The fatal event may be caused by abscess of the brain. For a full discussion of the subject the senior student is referred to the exhaustive monograph of Dr. Maude Abbott in ~~Vol. IV of~~ my "System of Medicine." I here abstract the conclusions of Hochsinger:

"(1) In childhood, loud, rough, musical heart murmurs, with normal or only slight increase in the heart dulness, occur only in congenital heart disease. The acquired endocardial defects with loud heart murmurs in young children are almost always associated with great increase in the heart dulness. In the transposition of the large arterial trunks there may be no cyanosis, no heart murmur, and an absence of hypertrophy.

"(2) In young children heart murmurs with great increase in the cardiac dulness and feeble apex beat suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

"(3) The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

"(1) An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which in early childhood is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses therefore an importance from a point of differential diagnosis which is not to be underestimated.

"(5) Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare except with congenital defects in the septum, and it speaks, therefore, against an acquired cardiac affection.

"(6) Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli, and can not be explained by the assumption of an endocarditis of the aortic valve."

Escherich suggests that the systolic basic murmur heard sometimes in the newborn, particularly if premature, may originate in the ductus Botalli before its closure.

Treatment.—The child should be warmly clad and guarded from all circumstances liable to excite bronchitis. In the attacks of urgent dyspnœa with lividity blood should be freely let. Saline cathartics are also useful. Digitalis must be used with care; it is sometimes beneficial in the later stages. When the compensation fails, the indications for treatment are those of valvular disease in adults.

VII. ANGINA PECTORIS

(*Stenocardia, Breast Pang*)

Definition.—A disease characterized by paroxysmal attacks of pain, usually pectoral, associated with changes in the vascular walls, organic or functional.

History.—In 1768 Heberden described a "disorder of the breast," to which he gave the name of "Angina Pectoris." Before this date Morgagni and Rougnon had described cases. The association with coronary artery disease was early shown by Jenner. John Hunter died in an attack. The connection with aortitis as demonstrated by Corrigan and Allbutt, the recognition of extra-pectoral forms, and the introduction of nitrites in treatment by Lauder Brunton are the important contributions of the nineteenth century.

Etiology.—The disease is not uncommon, about 700 dying yearly of it in England and Wales. In the United States it is more common, the average number of deaths per million of the population being more than double that of England.

It is a rare disease in hospitals; a case a year is about the average, even in the large metropolitan hospitals. It is a disease of the better classes, and a consultant in active work may see a dozen or more cases a year.

Age.—In my series of 268 cases there were, under 30, 9 cases; between 30 and 40, 41; between 40 and 50, 59; between 50 and 60, 81; between 60 and 70, 62; between 70 and 80, 13; above 80, 3. The fifth and sixth decades are the fatal periods, as shown by the Registrar General's statistics.

Sex.—Women are rarely attacked. Of my cases 231 were men and 37 women.

Race.—As mentioned, the disease seems to be relatively more frequent in the United States. Jews are particularly prone, 37 of my 268 cases.

Occupation.—It is not an affection of the working classes. The life of stress and strain, particularly of worry, seems to predispose to it, and this is perhaps why it is so common in our profession. In my series of 268 cases there were 33 physicians, a very large proportion. From John Hunter onward a long list of distinguished physicians have been its victims, among whom ~~may be mentioned~~ were in recent years Charcot, Nothnagel, and William Pepper.

Cardio-vascular Disease.—In persons under forty syphilis is an important feature, causing an aortitis, often limited to the root of the vessel. Whatever the cause, arterio-sclerosis predisposes to angina. A majority of the patients have sclerosis, many high blood pressure. Business men leading lives of great strain, and eating, drinking, and smoking to excess, form the large contingent of angina cases. Slight attacks may occur with high blood pressure alone.

Heredity.—The disease may occur in members of three generations, as in the Arnold family.

Imitative Features.—Outbreaks of angina-like attacks have been described. After the death of one member of a family from the disease, another may have somewhat similar attacks. Two of his physicians had angina after Senator Sumner's fatal attack. One of them died within two weeks; the other, a young man, recovered completely.

Symptoms. Gaged by the severity of the attacks, cases may be grouped in three categories:

(*a*) Mildest Form (*"Les Formes Frustes"* of the French).—There is a feeling of substernal tension, uneasiness, or distress, rising at times to positive pain, usually associated with emotion, sometimes with exertion, but soon passing off. There may be slight pallor, or a feeling of faintness. When rising to speak in public there may be a feeling of substernal tension—it is a common experience—which passes off. Muscular effort, as in climbing a hill or a stair, may bring on the sensation. In the high pressure life a man may experience for weeks or months this sense of substernal tension, not pain, and without accurate localization or radiation, and not increased by exercise or emotion. It is, as one patient expressed it, a "hot-box" indicating too great pressure and too high speed. It is away after the night's rest, and may disappear entirely when the "harness" is taken off.

(*b*) Mild Form (*Angina Minor*).—Pain in the heart of moderate severity with radiation to the arm is met with in many nervous and hysterical persons, in tobacco smokers, sometimes following the acute infections, particularly influenza. The attacks are rarely prolonged, are brought on by emotion, are more frequent in women, and are never fatal. Often called pseudo, false, functional, or toxic angina, the difference in the character of the attacks may be one of degree only. The conditions under which the attacks come on are of greater importance than the nature of the attack itself. There may be marked vaso-motor disturbance, with cold, numb, and blue extremities, followed by præcordial pain and a feeling of faintness. In persons addicted to tea, coffee, and tobacco heart pain is not infrequent, sharp and shooting, associated with palpitation, or severe and truly anginal.

(*c*) Severe Angina (*Angina Major*).—The two special features in this group are the existence in a large proportion of all the cases of organic disease of heart or vessels and the liability to sudden death. An exciting cause of the attack can usually be traced; muscular effort is the most common. Mental emotion is a second potent cause. John Hunter used to say that "his life was in the hands of any rascal who chose to worry him," and his fatal attack occurred in a fit of anger. A third very common excitant is flatulent distention of the stomach. Many patients are very sensitive to cold, and the chill of getting out of bed or of the bath may bring on a paroxysm.

Phenomena of the Attack.—During exertion or intense mental emotion the patient is seized with an agonizing pain in the region of the heart and a sense of constriction, as if the heart had been seized in a vice. The pains radiate to the neck and down the arm, and there may be numbness of the fingers or in the cardiac region. The face is usually pallid and may assume an ashy gray tint, and not infrequently a profuse sweat breaks out over the surface. The paroxysm lasts from several seconds to a minute or two, during which, in severe attacks, the patient feels as if death were imminent. As pointed out by Latham, there are two elements in it, the pain—*dolor pectoris*—and the indescribable feeling of anguish and sense of imminent dissolution—*angor animi*. There are great restlessness and anxiety, and the patient may drop dead at the height of the attack or faint and pass away in syncope. The condition of the heart during the attack is variable; the pulsations may be uniform and regular. The pulse tension, however, is usually increased, but it is surprising, even in the cases of extreme severity, how slightly the character of the pulse may be altered. After the attack there may be eructations, or the passage of a large quantity of clear urine. The patient usually feels exhausted, and for a day or two may be badly shaken; in other instances in an hour or two the patient feels himself again. While dyspnœa is not a constant feature, the paroxysm is not infrequently associated with a form of asthma; there is wheezing in the bronchial tubes, which may come on very rapidly, and the patient gets short of breath.

Death may occur in the first attack, as in the well known case of Thomas Arnold; or at the end of a series of attacks, the so-called *status angiosus*. Paroxysms may occur at intervals of a few weeks for a year or more before the fatal attack.

There is a chronic form represented by ten cases in ~~my~~ series, in which attacks occur irregularly. John Hunter's first seizure was in 1773, and he had many in the 20 years before his death. Sometimes life is a terrible burden, as any emotion or effort may bring on an attack. And, lastly, after paroxysms of great severity recurring for months, or even for so long as two years, as in one of ~~my~~ cases, complete recovery takes place.

Extra-pectoral Features of Angina.—In the attack the pain usually radiates up the neck and down the left arm. As the studies of Mackenzie and Head have shown in disease of the heart and of the aorta, the pain is referred to the 1st, 2d, 3d, and 4th dorsal areas; and in angina it may be also in areas of the distribution of the 5th to the 9th dorsal nerves. The pain may begin in the left arm, or in the jaw, even in the front teeth, or in one testis. Sometimes the pain remains in these distant parts, and yet the attack presents, as noted by Heberden, all the features of angina. ~~I have known~~

Bramwell. Edin. Med. J. Dec. 15.

1. Typical cases of pain left arm. absent in cases of left vent. anx. with atrd. between with atrd. undecided. sternum & left scapula.

2. Pain to rgt. arm alone. absent in cases of from rgt vent. hemorrhage over aorta

3. Both arms. - with atrd. both to rgt. clavicle sp. and.

In 45 cases q. 178 - aortic disease or aneurism.

the attack to begin with agonizing pain in the left leg; in another case in the left pectoral muscle. The entire features of the attack may be sub-diaphragmatic—the so-called *angina abdominis*. In at least ten of my series the pains were abdominal, and, as first pointed out by Leared, gastralgia may be diagnosed.

The *pulmonary features* of angina are remarkable. A condition like acute emphysema may come on, with wheezing and an inflated state of the lungs. Acute œdema may follow with the expectoration of large quantities of a thin, bloody fluid. The blood pressure may be extraordinarily high—340 mm. Hg. in one case. *Cerebral features* are not common, but unconsciousness may occur. Transient monoplegia, or hemiplegia and aphasia, occurred in three of my cases.

Morbid Anatomy and Pathology.—The 17 post mortems in my series illustrate the usual lesions:

(*a*) *Coronary artery disease* was present in 13 cases. The orifices only may be involved in a sclerotic aortitis. In one case they were narrowed to admit only a bristle, while the vessels beyond were normal. Blocking of a branch with a fresh thrombus, or with an embolus, is not uncommon. During an attack an infarct may soften, with perforation of the ventricular wall. Obliterative endarteritis, *the* lesion of the disease, was present in 9 of my cases. In elderly subjects the coronary vessels may be calcified—the condition found by Jenner in John Hunter.

(*b*) *Aortitis* was present in four of my cases, in syphilitic subjects, all under 40 years of age. Corrigan first called attention to this lesion in angina, the great importance of which has been emphasized by Clifford Allbutt. It is usually limited to the supra-sigmoidal area, and has the characteristic features of the syphilitic aortitis.

(*c*) In a few instances no lesions have been found. In one case of my list a man aged 26 had attacks, which were regarded as functional, on and off for two years. Death occurred after a series of paroxysms. The aorta was small, otherwise there were no changes.

No completely satisfactory explanation of the phenomena of the angina attack has yet been offered. It has been regarded as a neuralgia of the cardiac nerves, a cramp of the heart muscle, or of certain parts of it, or an expression of tension of the ventricular walls in extreme dilatation. In some ways the intermittent claudication theory of Allan Burns meets the case. This may be defined as a state in which an artery admits enough blood to a muscular structure for quiet work, but not enough for increased work, so that the contractile function of the muscle is disturbed and pain results. Burns remarked that "... If we can call into vigorous action a limb around which we have with moderate tightness applied a ligature, we find that then the member can only support its action for a very short time, for now the supply of energy and its expenditure do not balance each other. ... A heart, the coronary arteries of which are cartilaginous or ossified, is nearly in a similar condition; it can, like the limb begirt with a moderately tight ligature, discharge its functions so long as its action is moderate and equal. Increase, however, the action of the whole body, and along with the rest that of the heart, and you will soon see exemplified the truth of what has been said."

Angina results from an alteration in the working of the muscle fibres of any part of the cardio-vascular system, whereby painful afferent stimuli are excited. Cold, emotion, or toxic agents interfering with the orderly action of the peripheral circulation increase the tension in the heart walls or in the larger central mains, causing strain and a type of contraction capable of exciting in the involuntary muscles painful afferent stimuli. In disturbance of this all-important Gaskellian function, in the stretching, in the alteration of the wall tension at any point, sufficient to excite a pain-producing resistance to this by the muscle elements, are to be sought an explanation of the phenomenon of the attack. Spasm, or narrowing of a coronary artery, or of one branch, may so modify the action of a section of the heart that it works with disturbed tension, and with stretching and strain sufficient to rouse painful sensations. Or the heart may be in the same state as the leg muscles of a man with intermittent claudication, working smoothly when quiet, but the instant an effort is made, or if a wave of emotion touches the peripheral vessels, the normal contraction is disturbed and a crisis of pain excited.

There are three modes of dying in angina—one, as Walshe says, "is sudden, instantaneous, coeval with a single pang." The functions of life stop abruptly, and with a gasp all is over. In a second mode, following a series of attacks, the heart grows weaker and the patient dies in a progressive asthenia; while in a third there is a gradually induced cardiac insufficiency with dyspnœa.

Prognosis.—In men under 40 syphilis must be suspected, and with appropriate treatment recovery may be complete (see ~~my~~ Lumleian Lectures, Lancet, 1910, I). In men in the 5th and 6th decades who have lived the high pressure life a change of habits may bring relief; but, as Walshe remarked, "the cardinal fact in real angina is its uncertainty." Even after attacks of the greatest severity recovery is possible. The circumstances that bring on an attack are important. Emotion is of the least importance. The angina of effort that follows any slight exertion is, as a rule, more serious than that which comes on spontaneously, or is excited by emotion; yet one of ~~my~~ patients who could never dress without having what he called "angor de toilette" lived for 11 years. The cardio-vascular condition is of the first importance in prognosis. Very high blood pressure, advanced arterio-sclerosis, valvular disease, signs of myocardial weakness are of serious import. It is to be remembered that a large proportion of all cases have no obvious signs of cardiac disease; and the coronary arteries may be extensively diseased with clear heart sounds and a good pulse. In women the forms of angina with marked vaso-motor disturbance as a rule do well, and when neurotic or hysterical manifestations are prominent the outlook is good.

Treatment.—Syphilitic cases require active treatment—~~salvarsan~~ in the subjects under 40, mercury and iodide of potassium in older persons. In the neurotic cases with a recognition of the basic disturbance in the vaso-motor system a Weir Mitchell cure and hydrotherapy are indicated. A persistent course of wet packs ~~I have~~ often ~~seen~~ helpful. When high tension is present, as is not infrequently the case ~~in neurasthenia~~, the nitrites may be given; and ergotin grs. ii (0.13 gm.) three times a day has a definite value in vaso-motor instability. In the severer types ~~of the disease~~ the treatment is

Suggested that sudden death may be from ventricular fibrillation.
"Heterotropic" impulses.

concerned with the attack and with the general condition afterward. In the attack inhalation of nitrite of amyl, introduced by Lauder Brunton, may give instant relief. We see its benefit particularly in cases with widespread arterial constriction. In the recurring terrible paroxysms it may lose its effect, but many milder forms are relieved promptly, and it gives great comfort and confidence to the patient to carry the *perles*. Morphia should be used freely when amyl nitrite fails and when the attacks recur with great frequency. As Burney Yeo pointed out, angina patients are very resistant to this drug, and between 10 p. m. and 1 p. m. the next day I have known five grains administered with relief to the pain, but without causing sleep. Chloroform may have to be used, and my experience coincides with that of the late George Balfour of Edinburgh, that it is always helpful, never harmful. With a dusky cyanosis and asthma like breathing oxygen inhalations may be given.

For the general condition, if high tension is present, iodide of potassium and the nitrites in all forms are useful. The use of theobromine has been warmly advocated by Marchiafava, grs. xv (1 gm.) three times a day, and I have tried it in a few cases with benefit, particularly in two cases of the angina of effort.

C. DISEASES OF THE ARTERIES

I. ARTERIO-SCLEROSIS

(*Arterio-capillary Fibrosis*)

The conception of arterio-sclerosis as an independent affection—a general disease of the vascular system—is due to Gull and Sutton.

Definition.—A condition of thickening of the arterial coats, with degeneration, diffuse or circumscribed. The process leads, in the larger arteries, to what is known as atheroma and to endarteritis deformans, and seriously interferes with the normal functions of various organs.

Etiology.—Among the important factors in causing arterio-sclerosis the following may be considered:

(*a*) Hypertension.—The blood pressure, the tension or force with which the blood circulates, depends upon five factors: The heart pump supplies the force; the elastic coats of the large arteries store and convert an intermittent into a continuous stream; the small arteries act as sluices or taps regulating the control to different parts; the capillary bed is the irrigation field over which the nutritive fluid is distributed; and the drainage system is represented by the veins and lymph channels.

Galen first grasped the fact that life depends upon the maintenance of a due pressure in these irrigation fields: "Many canals dispersed throughout all the parts of the body convey to them blood as those of a garden convey moisture, and the intervals separating those canals are wonderfully disposed by nature in such a way that they should neither lack a sufficient quantity of blood for absorption, nor be overloaded at any time with an excessive supply."

The blood pressure varies greatly in different individuals, and in the same individual under varying conditions. The normal blood pressure is from 120 to 130 mm. of mercury, but in persons over 50 it is very often from 110 to 160 mm. A permanent pressure above the latter figure may be called high, but there are great regional variations. Permanently low blood pressure may be met with in asthenia from any cause, in the various toxæmias of the infectious diseases, and there are persons in apparently good health with chronic hypotension.

High tension is met with in many chronic diseases, in various forms of cardiac and renal disease, in lead poisoning, and, above all, in connection with general arterio-sclerosis. The relation to arterio-sclerosis has been much discussed. Briefly, there are three groups of cases: (1) First, the simple high tension without signs of arterial or renal disease—what Clifford Allbutt calls hyperpyesis. In this well recognized condition, met with in individuals otherwise healthy, the blood pressure is permanently high—above 180—but, so far as can be ascertained, there are no arterial, cardiac, or renal changes. It is difficult, of course, to exclude internal, not discernible alterations in the splanchnic and other vessels, since, as is well known, vascular disease may be very localized. But clinically the group is well defined and very important. The condition is met with most frequently in keen business men, who work hard, drink hard, and smoke hard.

The exact cause of this high tension we do not know. Some have attributed it to over-activity of the adrenals, but it is much more likely that the primary difficulty is somewhere in the capillary bed—in that short space in which the real business of life is transacted. However produced, the important point here is that this hypertension itself leads to arterio-sclerosis, which can be produced experimentally by the injection of adrenalin and other hypertensive substances.

(2) In the second group of cases the high tension is associated with an arterio-sclerosis with consecutive cardiac and renal disease.

(3) In the third group the high tension is secondary to forms of chronic nephritis in association with cardio-vascular disease.

(*b*) As an INVOLUTION PROCESS arterio-sclerosis is an accompaniment of old age, and is the expression of the natural wear and tear to which the tubes are subjected. Longevity is a vascular question, which has been well expressed in the axiom that "a man is only as old as his arteries." To a majority of men death comes primarily or secondarily through this portal. The onset of what may be called physiological arterio-sclerosis depends, in the first place, upon the quality of arterial tissue (vital rubber) which the individual has inherited, and secondly upon the amount of wear and tear to which he has subjected it. That the former plays a most important rôle is shown in the cases in which arterio-sclerosis sets in early in life in individuals in whom none of the recognized etiological factors can be found. Thus, for instance, a man of twenty-eight or twenty-nine may have the arteries of a man of sixty, and a man of forty may present vessels as much degenerated as they should be at eighty. Entire families sometimes show this tendency to early arterio-sclerosis—a tendency which can not be explained in any other way than that in the makeup of the machine bad material was used for the

[illegible]

There are

tubing. More commonly the arterio-sclerosis results from the bad use of good vessels.

(*c*) ~~Chronic~~ Intoxications.—Alcohol, lead, and gout play an important rôle in the causation of arterio-sclerosis, although the precise mode of their action is not yet very clear. They may act, as Traube suggests, by increasing the peripheral resistance in the smaller vessels and in this way raising the blood tension, or possibly, as Bright taught, they alter the quality of the blood and render more difficult its passage through the capillaries. The observations of Cabot have thrown doubt on the importance of alcohol as a factor.

The poisons of the acute infections may produce degenerative changes in the media and adventitia. Thayer ~~has recently~~ called attention to the frequency of arterial changes as a sequence of typhoid fever.

(*d*) Syphilis, one of the most important single causes, will be spoken of under morbid anatomy.

(*e*) Overeating.—I am more and more impressed with the part played by overeating in inducing arterio-sclerosis. There are many cases in which there is no other factor. George Cheyne's advice, quoted at page ~~451~~, was never more needed than by the present generation.

(*f*) The Stress and Strain of Modern Life.—There are men in the fifth decade who have not had syphilis or gout, who have eaten and drunk with discretion, and in whom none of the ordinary factors are present—men in whom the arterio-sclerosis seems to come on as a direct result of a high pressure life.

(*g*) Overwork of the muscles, which acts by increasing the peripheral resistance and by raising the blood pressure.

(*h*) Renal Disease.—The relation between the arterial and kidney lesions has been much discussed, some regarding the arterial degeneration as secondary, others as primary. There are two groups of cases, one in which the arterio-sclerosis is the first change, and the other in which it is secondary to a primary affection of the kidneys.

Morbid Anatomy.—The affection is met with most frequently in the aorta and its main branches. It is comparatively less frequent in the mesenteric and rare in the pulmonary arteries. Several different forms ~~may be recognized~~:

(*a*) Nodular.—The aorta presents in the early stages, from the ring to the bifurcation, numerous flat projections, yellowish or yellowish white in color, and situated particularly about the orifices of the branches. In the early stage these patches are scattered and do not involve the entire intima. In more advanced stages the patches undergo atheromatous changes. The material constituting the button undergoes softening and breaks up into granular material, consisting of molecular débris—the so-called atheromatous abscess.

(*b*) Diffuse Arterio-sclerosis.—In this form, met with usually in middle-aged men, or younger persons, the affection is widespread throughout the arteries. In the aorta the media shows necrotic and hyaline changes, while the intima may be smooth or show very slight thickenings—scattered elevated areas of an opaque white color, some of which undergo atheromatous changes. The smaller arteries show thickening of the walls, due particularly

to increase in the sub-endothelial connective tissue. The muscular coat may be at first hypertrophied, but later undergoes hyaline and calcareous changes. In this group of cases the heart hypertrophies and later fibrous myocarditis is often present. The semilunar valves are opaque and sclerotic. The kidneys are sclerotic, and may be increased in size, and are usually very firm. In places the surface may be rough, or present atrophied depressed areas of a deep red color.

(*c*) SENILE ARTERIO-SCLEROSIS.—In this the larger arteries are dilated and tortuous, the walls thin and stiff, and the smaller vessels, as the radials, converted into rigid tubes like pipe-stems. The intima of the aorta may be occupied by rough, calcareous plaques, with here and there fissures and loss of substance. There may be sub-endothelial softening with the formation of atheromatous ulcers on which thrombi may deposit; though, as a rule, there may be the most extreme calcification and roughness with erosions of the aorta without thrombus formation. In the smaller vessels, as the radicals, there are degeneration and calcification of the media—the so-called Monckeberg type.

The SYPHILITIC ARTERIO-SCLEROSIS is usually a mesaortitis with definite characteristics. Macroscopically it may be limited in extent, localized at the root of the aorta, or about the orifice of an aneurism, or there is a band of an inch in width on some portion of the tube, while other parts of the aorta and its branches are normal. In other instances the intima is involved, not with the usual plaque-like areas of atheroma, but there are shallow depressions of a bluish tint, and short transverse or longitudinal puckerings, sometimes with a stellate arrangement; or the intima is pitted and scarred with small depressions and linear sulci. Microscopically the most important changes are found in the media and adventitia: (*a*) perivascular infiltration of the vasa vasorum; (*b*) small-celled infiltration in areas of the media, with (*c*) splitting, separation, and destruction of elastic fibres and the muscle cells. The intima over these areas may be perfectly normal, but it often shows signs of thickening with fatty degeneration and the production of hyaline. Similar changes have been described by Klotz in the larger blood vessels in cases of congenital syphilis. And, lastly, the specific nature of this mesaortitis has been determined by the detection of the spirochætes. Other forms affecting the smaller vessels have been referred to under syphilis.

SCLEROSIS OF THE PULMONARY ARTERY is met with in all conditions which for a long time increase the tension in the lesser circulation, particularly in mitral valve disease and in emphysema. Sometimes the sclerosis reaches a high grade and is accompanied with aneurismal dilatation of the primary and secondary branches, more rarely with insufficiency of the pulmonary valve. Leonard Rogers has shown that in India it is not uncommon as a primary affection. In a remarkable case of a young man of twenty four, reported by Romberg from Curschmann's clinic, the pulmonary arteries were involved in most extensive arterio-sclerosis; the main branches were dilated, and the smaller branches were the seat of the most extreme sclerotic changes. On the other hand, the aorta and its branches were normal.

In many cases of arterio-sclerosis the condition is not confined to the arteries, but extends not only to the capillaries but also to the veins, and may properly be termed an *angio-sclerosis*.

Arteriitis syphilitica obliterans. – Ruhe, Mesantin +
Massage, Bäder, Elektrisieren.
Occlusion des ganzen Gefäßsystems. – Lebtag
Carotis mit Thrombose – (auch Carotis + subclavia affected)

Art. sclerosis

of pulmonary artery. usually in young.

symptoms. angina. mitral insufficiency. hypertrophy of rt. vent. rare to sclerosis pul. veins found narrow in some cases

—

Anatomy similar with high bp. —

some cases - high rbc count - looked like polycythaemia esp. if emphysema - chronic bronchitis

In cerebral group - always look for hypertension

Sclerosis of the veins—*phlebo-sclerosis*—is not at all an uncommon accompaniment of arterio-sclerosis. It is seen in conditions of heightened blood pressure, as in the portal system in cirrhosis of the liver and in the pulmonary veins in mitral stenosis. The affected vessels are usually dilated, and the intima shows, as in the arteries, a compensatory thickening, which is particularly marked in those regions in which the media is thinned. The new-formed tissue in the endophlebitis may undergo hyaline degeneration, and is sometimes extensively calcified. Without existing arterio-sclerosis the peripheral veins may be sclerotic, usually in conditions of debility, but not infrequently in young persons.

Symptoms.—Increased Tension.—The pressure with which the blood flows in the arteries depends upon the degree of peripheral resistance and the force of the ventricular contraction. A high-tension pulse may exist with very little arterio-sclerosis; but, as a rule, when the condition has been persistent, the sclerosis and high tension are found together. On the other hand, a very low or normal tension may be present in extremely sclerotic vessels.

General Symptoms.—The early symptoms are interesting. Stengel has called attention to the pallor, and there may be dyspeptic symptoms. It is remarkable with what rapidity the disease may progress. I have known the peripheral arteries to stiffen and grow old in a couple of years.

The combination of heightened blood pressure, a palpable thickening of the arteries, hypertrophy of the left ventricle, and accentuation of the aortic second sound are signs pathognomonic of arterio-sclerosis. From this period of establishment the course of the disease may be very varied. For years the patient may have good health, and be in a condition analogous to that of a person with a well compensated valvular lesion. There may be no renal symptoms, or there may be the passage of a larger amount of urine than normal, with transient albuminuria, and now and then hyaline tube casts. The subsequent history is extraordinarily diverse, depending upon the vascular territory in which the sclerosis is most advanced, or upon the accidents which are so liable to happen, and the symptoms may be cardiac, cerebral, renal, etc.

(*a*) *Cardiac.*—The involvement of the coronary arteries may lead to the various symptoms already referred to under that section—thrombosis with sudden death, fibroid degeneration of the heart, aneurism of the heart, rupture, and angina pectoris. Angina pectoris is not uncommon, and the organic variety is almost always associated with arterio-sclerosis. A second important group of cardiac symptoms results from the dilatation which finally gets the better of the hypertrophy. The patient then presents all the symptoms of cardiac insufficiency—dyspnœa, scanty urine, and very often serous effusions. If the case has come under observation for the first time the clinical picture is that of chronic valvular disease, and the existence of a loud blowing murmur at the apex may throw the practitioner off his guard. Many cases terminate in this way.

(*b*) The *cerebral* symptoms of arterio-sclerosis are varied and important, and embrace those of many degenerative diseases, acute and chronic (which follow sclerosis of the smaller branches), and cerebral hæmorrhage.

Transient hemiplegia, monoplegia, or aphasia may occur in advanced arterio-sclerosis. The attacks are very characteristic, often brief, lasting twenty

four hours or less. Recovery may be perfect. Recurrence is the rule, and a patient may have a score or more attacks of aphasia, or in the course of a couple of years there may be half a dozen transient hemiplegic attacks or one or two monoplegias, or paraplegia for a day or two. Much attention has of late been given to these cases, which seem best explained on the view of transient spasm as originally suggested by Peabody. Vertigo occurs frequently, and may be either simple, or in association with slow pulse and syncopal or epileptiform attacks—the Stokes-Adams syndrome.

(*c*) *Renal* symptoms supervene in a large number of the cases. A sclerosis, patchy or diffuse, is present in a majority of the cases at the time of autopsy, and the condition is practically that of contracted kidney. It is seen in a typical manner in the senile form, and not infrequently develops early in life as a direct sequence of the diffuse variety. It is often difficult to decide clinically (and the question is one upon which good observers might not agree in a given case) whether the arterial or the renal disease has been primary.

(*d*) *Abdominal Arterio-sclerosis.*—It is believed to be associated particularly with overeating and chronic overtaxing of the stomach and intestines with work. The condition is by no means uncommon, and the sclerosis of the splanchnic vessels may be advanced out of all proportion to that elsewhere. The symptoms are indefinite, sometimes resembling those of the ordinary neurosis with marked constipation, features that are by no means certainly associated with sclerosis; on the other hand, there is much more reason to connect the attacks of severe abdominal pain, the gastric crises of lead and of tabes with spasm of the vessels in this condition. There are cases of angina pectoris with abdominal pain which may be due to angiospasm of the sclerotic vessels.

(*e*) Among other events in arterio-sclerosis may be mentioned gangrene of the extremities, due either directly to endarteritis or to the dislodgment of thrombi. Sudden transient paralysis may occur.

(*f*) *Intermittent lameness or claudication*, the dysbasia angio-sclerotica of Erb, the crural angina of Walton, is seen most frequently in connection with arterio-sclerosis. In the horse, in which the intermittent lameness was first described by Bouley, verminous aneurisms are present in the iliac arteries. In man Charcot described the condition in 1856 in an old soldier who was not able to walk for more than a quarter of an hour without severe cramps in the legs. The post mortem showed a traumatic aneurism of one iliac artery. The loss of function and the pain in the muscles were due to the relative ischemia. Erb has shown that intermittent lameness is not at all infrequent, particularly among private patients, only 2 of his 45 cases not coming in this class. Of 127 cases there were only 7 in women. Hebrews seem more frequently affected. Syphilis, alcohol, and tobacco are common factors. Muscular weakness after exertion or complete disability, numbness, tingling, and paræsthesia of various forms are the common symptoms. Pulsation may be absent in the dorsal arteries of the feet and the vessels are sclerotic. Vaso-motor changes may be present, and in the dependent position the feet and legs become deeply congested.

Treatment.—In the late stages the conditions must be treated as they arise in connection with the various viscera. In the early stages, before any

local symptoms are manifest, the patient should be enjoined to live a quiet, well regulated life, avoiding excesses in food and drink. It is usually best to explain frankly the condition of affairs, and so gain his intelligent coöperation. Special attention should be paid to the state of the bowels and urine, and the secretion of the skin should be kept active by daily baths. Alcohol in all forms should be prohibited, and the food should be restricted to plain, wholesome articles. The use of mineral waters or a residence every year at one of the mineral springs is usually serviceable. If there has been a syphilitic history the persistent use of iodide of potassium is indicated; indeed, even in the non-syphilitic cases it seems to do good. It is best given in small doses, grains v to x (0.3 to 0.6 gm.). Whenever the blood pressure is high nitroglycerin or the sodium nitrite may be given to relieve symptoms rather than with any hope of essentially influencing the disease.

In cases which come under observation for the first time with dyspnœa, slight lividity, and signs of cardiac insufficiency, venesection is indicated. In some instances, with very high tension, striking relief is afforded by the abstraction of 10 to 20 ounces of blood. Cardiac failure, renal symptoms, etc., require the usual treatment.

II. ANEURISM

Definition.—A tumor containing fluid or solid blood in direct communication with the cavity of the heart, the surface of a valve, or the lumen of an artery.

History.—Galen knew external aneurism well, and in the second century A. D., Antyllos devised his operation of incising and emptying the sac inclosed between ligatures. Internal aneurism was recognized by Fernelius in the 16th century, and Vesalius was very familiar with the disease. Ambroise Paré suggested the relation of aneurism to syphilis, which was insisted upon in the great monograph of Lancisi in 1728. Morgagni in 1761 described very fully the symptoms and morbid anatomy. The modern views date from the studies of Helmstedter and Köster, who showed that the primary change was in the media. The researches of Eppinger, Thoma, and Welch emphasized the importance of these changes in the media, particularly as brought about by syphilis.

Classification.—For practical purposes the following classification may be adopted:

I. True aneurism (aneurisma verum or aneurisma spontaneum), in which one or more of the coats of the vessel form the wall of the tumor: (*a*) *Dilatation-aneurism*—(1) Limited to a certain portion of the vessel, fusiform, cylindroid; (2) extending over a whole artery and its branches—cirsoid aneurism. (*b*) *Circumscribed saccular aneurism,* which is the common form of aneurism of the aorta. (*c*) *Dissecting aneurism,* with splitting of the media, and occasionally with the formation of a new tube lined with intimal endothelium.

II. False aneurism, following a wound or the rupture of an artery, or of a true aneurism, causing a diffuse, or circumscribed, hæmatoma.

III. Arterio-venous aneurism, either with direct communication between an artery and vein, or with the intervention of a sac, varicose aneurism.

IV. Special forms, such as the parasitic, the erosion, the traction, the mycotic.

Etiology.—Predisposing Causes.—*Age.*—Nearly one half of the deaths in England and Wales from aneurism in males occur between the ages of 30 and 45. In the young and in the very old the disease is rare, but it may occur at any age. Congenital aneurism has been described.

Sex.—Males are attacked much more frequently than females—in a ratio of 5 to 1.

Race and Locality.—The disease is more common in Great Britain than on the Continent. Among about 19,000 post mortems at Vienna there were 230 cases of aneurism, while among 18,678 at Guy's Hospital there were 325 cases. It is more common in the negroes of the Southern States of America than among the whites. Of 345 admissions for aneurism to ~~my~~ wards at the Johns Hopkins Hospital 132 were in colored and 213 in white patients—a ratio of 1 to 1.6, while the ratio of white to colored in the hospital at large was 5 to 1. In India aneurism is rare, though syphilis and arterial disease are common. Possibly, as Rogers suggests, the low blood pressure in the natives may have something to do with this comparative immunity.

Occupation.—Soldiers, sailors, draymen, iron and steel workers, and dock workers are particularly prone. In soldiers and sailors, who are peculiarly liable, the disease ~~appears to be~~ in direct proportion to the prevalence of syphilis.

Determining Causes.—These are three in number:

I. *The Acute Infections.*—In the specific fevers areas of degeneration are common in the aorta. Fortunately in most instances they are confined to the intima, but occasionally, as Thayer ~~has~~ pointed out in typhoid fever, the changes may be in the media. *The* infection with which aneurism is especially connected is syphilis—a fact recognized in the eighteenth century by Lancisi and by Morgagni, and dwelt upon specially in 1876 by Francis H. Welch, of the British Army. All recent figures show a very high percentage of syphilis in the subjects of aneurism—as high as 80 or 85 per cent.; and nowadays it is rare not to find a positive Wassermann reaction in an aneurismal patient under fifty. The lesion, a mesaortitis, has been described under arterio-sclerosis.

Other infections play a very minor rôle in the disease. With rheumatic fever, pneumonia, and septicæmia, the mycotic aneurism may be associated.

The various toxic factors which favor arterial degeneration, such as alcohol, lead, tobacco, and the chronic endogenous toxæmias, as gout and ~~Bright's disease~~, are rare determining causes.

II. The second determining factor is *strain,* particularly the internal strain associated with sudden and violent muscular effort. The media is the protecting coat of the artery, and during a violent effort, as in lifting or jumping, laceration or splitting of the intima may occur over a weak spot. If small this leads to a local bulging of the media and the gradual production of a sac, ~~or~~ the tear of the intima may heal completely, or a dissecting aneurism may form. In other instances a widespread mesaortitis leads to a gradual, diffuse distention of the artery. This type of aneurism, frequently seen in the aged, may follow ordinary chronic atheroma.

III. *Occasional Causes.*—(*a*) Embolism: The emboli may consist of

Crisp: Brun-

Aortitis - syphilitic - N.Y. State Jan. 9 1905. Dec-15 - Lancet Nov.

vegetations or calcified fragments from the valves. This form of aneurism, often multiple, is met with in infective endocarditis, as in the remarkable case which was described by me in 1888. In infective endocarditis the emboli probably pass to the vasa vasorum, causing mesaortitis with weakening of the wall; but in the smaller vessels the aneurisms are caused by the direct lodgment of the emboli which infect and weaken the wall. (*b*) External Injury: A blow on the chest, a sudden fall, or the jar of an accident may cause a rupture of the intima over a weak spot in the aorta, with the production of a dissecting or sacculated aneurism. (*c*) External Erosion: A tuberculous focus may involve the wall of the aorta; or a bullet lodged near the wall of an artery may weaken it and be followed by aneurism. (*d*) In the horse there is a *parasitic aneurism* common in the mesenteric vessels, due to growth in them of the *Strongylus armatus*. (*e*) Thoma has described a "traction" aneurism at the concavity of the arch at the point of insertion of the ductus Botalli.

Morbid Anatomy and Pathology.—Number.—Usually there is one aneurism, but three or four or even a dozen may be present. Multiple cup-shaped tumors in the aorta are always syphilitic. The mycotic are usually multiple, and in the peripheral vessels there may be a dozen or more.

Form.—There are two great types—one in which the lumen of the vessel is dilated, and the other in which a limited section of the wall gives way with the formation of a sac. Typical cylindrical and spindle shaped aneurisms are seen in the aorta and in the vessels of the second and third dimensions. The sacculated form is the more common. They are either flat, saucer-shaped, or cup-shaped, or sometimes beyond a very narrow orifice is a cylindrical tumor of variable size, from a pin's head in the smaller vessels, as in the brain, to a huge sac which may fill one half of the chest.

Vessels Affected.—Of a series of 501 cases studied by Crisp, the thoracic aorta was involved in 125, the abdominal aorta in 59, the femoral-iliac in 66, the popliteal in 137, the innominate in 20, the carotids in 25, subclavians in 23, axillary in 18. The other smaller vessels are rarely attacked. Of late years aneurism of the external vessels appears to have become much less frequent.

ANEURISM OF THE THORACIC AORTA

I. Dilatation Aneurism.—New interest has been attached to this form since the introduction of the X-rays in diagnosis. Formerly it was very often overlooked. The shape may be a single fusiform, or tent-shaped, or there may be multiple spindles. The condition was accurately noted by Hodgson, who called it "a preternatural permanent enlargement of the cavity of an artery," and distinguished it clearly from ordinary aneurism. It is very often associated with insufficiency of the aortic valves—a combination to which the French gave the name "Maladie de Hodgson." It is more common in elderly people, and may follow diffuse arterio-sclerosis from any cause. In syphilitic subjects it may be limited to the ascending portion of the arch, or may involve the entire arch.

Symptoms.—The cases are often latent, met with accidentally in medico-legal work, and the dilatation may reach an extreme grade without any symptoms. In other cases, particularly in the syphilitic aortitis in men, angina

pectoris is an early symptom, and dilatation may be present in the senile form. In a third group the features are those of organic disease of the heart, usually of aortic insufficiency. Of 60 cases analyzed by T. McCrae there were 18 patients under 40; the majority of the cases were in elderly persons. Pressure effects are not uncommon; the trachea may be flattened and the œsophagus compressed. Erosion of the bones is rare.

The PHYSICAL SIGNS are often characteristic. Visible pulsation in the episternal notch is usually present, and it may even extend to the right sterno-clavicular articulation. Pulsation may sometimes be seen in the first and second right interspaces. In about one half of the cases a diffuse pulsation over the manubrium is present, but in old persons with rigid chest walls there may be extreme dilatation without any visible impulse. A rough thrill is not infrequently present, or a diastolic thrill when the valves are insufficient. A sharp diastolic shock may be felt.

Dulness over the manubrium is the most constant single sign. The second sound may be of a clanging, metallic, or even amphoric quality. When a diastolic murmur is present it may be heard loudly over the manubrium and perhaps transmitted into the vessels of the neck. The blood pressure is often low—below 140 mm. in 35 of 40 cases studied by McCrae. Examination with the X-rays in skilled hands is the most satisfactory means of diagnosis, as the dilated aorta casts a very definite shadow, much larger than the normal aorta, and showing very little difference in extent during systole and diastole.

II. Dissecting Aneurism.—The majority of aneurisms of the aorta begin with a split or crack of the intima over a spot of syphilitic mesaortitis. Once this split has started the aorta may rupture in all its coats, or an aneurism may form at the site, or the fracture of the intima, though large and often circumferential, may heal; or the blood may extend between the coats, separating them for many inches, or in the entire extent, forming a dissecting aneurism; and, lastly, such a dissecting aneurism may heal perfectly.

RUPTURE OF THE AORTA is not very infrequent, as medico-legal work indicates. Usually there is agonizing pain with features of shock, and death may take place instantly; but in fully half of the cases there are two very characteristic stages, the first corresponding to the rupture of the inner coats, the second eight to ten hours, or as long as fifteen or sixteen days later, to fatal rupture of the external layer.

Dissecting aneurism is not very common. There were only two cases in 16 years at the Johns Hopkins Hospital, where aneurism may be said to be exceptionally frequent. The primary split is most frequently in the arch, not far above the valves, and is in the form of a transverse, or vertical, clean cut incision, as if made with a razor. The extent of the separation of the coats is variable. If the adventitia is reached, rupture is certain to take place, as only the structures of the middle coat can resist for any time the pressure of the blood. The blood may pass for three or four or more inches, separating the media, and then burst internally or externally. In other cases the dissection reaches from the ascending arch to the bifurcation of the aorta, even passing down the iliac and femorals into the smaller vessels of the leg. The splitting of the coats may, indeed, as in a case described by Rokitansky, reach to all the subdivisions of the aorta. The symptoms are

those spoken of under rupture; but a very remarkable condition may follow, leading to:

Healed Dissecting Aneurism.—The earlier observers of this remarkable condition regarded it as an anatomical anomaly of a double aorta. Adami has collected 39 cases, in a majority of which there was no advanced disease of the aorta itself. The outer tube formed by the dissecting aneurism may extend the entire length of the aorta, occupying the full extent of the circumference. The most extraordinary feature is that the outer tube may present a perfectly smooth and natural appearance, and be lined with a new intima. The condition may last for many years.

~~III. Sacculated Aneurism of the Aorta.~~ Aneurism of the Arch.—For purposes of discussion this part of the vessel may be divided into the sinuses of Valsalva, ascending, transverse, and descending portions.

(*a*) Aneurism of the sinuses of Valsalva, a common and important variety, is met with most frequently in young syphilitic subjects. There may be pouching of one or of all three sinuses; the aortic ring is apt to be involved and one or more of the valves rendered incompetent. The special features may be thus summarized: (1) It is often latent, causing sudden death by perforation into the pericardium. (2) It is a medico-legal aneurism met with most frequently in coroner's cases. (3) Angina pectoris is not uncommon and may be the only symptom. (4) Aortic insufficiency is often associated with it. (5) In a majority of all cases syphilitic mesaortitis is present.

(*b*) Aneurism of the Ascending Arch.—Along the convex border aneurism frequently arises and may grow to a large size, either passing out into the right pleura or forward, pointing at the second or third interspace, eroding the ribs and sternum, and producing large external tumors. In this situation the sac may compress the superior vena cava, causing engorgement of the vessels of the head and arm; sometimes it compresses only the subclavian vein, and causes enlargement and œdema of the right arm. Perforation may take place into the superior vena cava, of which accident Pepper and Griffith ~~have~~ collected 29 cases. In rare instances, when the aneurism springs from the concave side of the vessels, the tumor may appear to the left of the sternum. Large aneurisms in this situation may cause much dislocation of the heart, pushing it down and to the left, and sometimes compressing the inferior vena cava, and causing swelling of the feet and ascites. The right recurrent laryngeal nerve is often compressed. The innominate artery is rarely involved. Death commonly follows from rupture into the pericardium, the pleura, or into the superior cava; less commonly from rupture externally, sometimes from syncope.

(*c*) Aneurism of the Transverse Arch.—The direction of growth is most commonly backward, but the sac may grow forward, erode the sternum, and form a large tumor. The sac presents in the middle line and to the right of the sternum much more often than to the left, which occurred in only 4 of 35 aneurisms in this situation (O. A. Browne). Even when small and producing no external tumor it may cause marked pressure signs in its growth backward toward the spine, involving the trachea and the œsophagus, and giving rise to cough, which is often of a paroxysmal character, and dysphagia. The left recurrent laryngeal is often involved in its course round

the arch. A small aneurism from the lower or posterior wall of the arch may compress a bronchus, inducing bronchorrhœa, gradual bronchiectasis, and suppuration in the lung—a process which by no means infrequently causes death in aneurism, and a condition which at the Montreal General Hospital we were in the habit of terming "aneurismal phthisis." Occasionally enormous aneurisms arise in this situation, and grow into both pleuræ, extending between the manubrium and the vertebræ; they may persist for years. The sac may be evident at the sternal notch. The innominate artery, less commonly the left carotid and subclavian, may be involved in the sac, and the radial or carotid pulse may be absent or retarded. Pressure on the sympathetic may at first cause dilatation and subsequently contraction of the pupil. Sometimes the thoracic duct is compressed.

The ascending and transverse portions of the arch are not infrequently involved together, usually without the branches; the tumor grows upward, or upward and to the right.

(*d*) Aneurism of the Descending Portion of the Arch.—It is not infrequently the traction aneurism of Thoma. The sac projects to the left and backward, and often erodes the vertebræ from the third to the sixth dorsal, causing great pain and sometimes compression of the spinal cord. Dysphagia is common. Pressure on a bronchus may induce bronchiectasis, with retention of secretions, and fever. A tumor may appear externally in the region of the scapula, and here attain an enormous size. Death not infrequently occurs from rupture into the pleura, or the sac may grow into the lung and cause hæmoptysis.

(*e*) Aneurism of the Descending Thoracic Aorta.—This is the least common situation of aortic aneurism. The larger number occur close to the diaphragm, the sac lying upon or to the left of the bodies of the lower dorsal vertebræ, which are often eroded. It is frequently latent, in 3 of 14 cases reported by me, and is often overlooked; pulmonary and pleural symptoms are common. Pain in the back is severe; dysphagia is not infrequent. The sac may reach an enormous size and form a subcutaneous tumor in the left back.

Physical Signs.—Inspection.—A good light is essential; cases are often overlooked owing to a hasty inspection. The face is often suffused, the conjunctivæ injected, and veins of the chest and of one arm engorged. One pupil may be enlarged. In many instances inspection is negative. On either side of the sternum there may be abnormal pulsation, due to dislocation of the heart, to deformity of the thorax, or to retraction of the lung. Three sorts of pulsation may be seen in the chest: (1) A general shock, such as is seen in the violent throbbing of the heart or of an aneurism. In anæmia, in neurasthenia, and in great hypertrophy this widespread shock may suggest aneurism. (2) A diffuse impulse localized in a certain part of the chest, which may be caused by a deep-seated aneurism but which is met with also in tumors, in pulsating pleurisy, and in a few cases without evident cause (see "Modern Medicine," Vol. IV, p. 474). (3) The punctate, heaving true aneurismal impulse which when of any extent is visibly expansile. It is seen most frequently above the level of the third rib to the right of the sternum, in the second left interspace, over the manubrium, and behind in the left interscapular region. When the innominate is involved the throbbing may

Thou may be

be seen at the right sterno-clavicular joint and above it. An external tumor is present in many cases, projecting either through the upper part of the sternum or to the right, sometimes involving the sternum and costal cartilages on both sides, forming a swelling the size of a cocoanut or even larger. The skin is thin, often blood stained, or it may have ruptured, exposing the laminæ of the sac. The apex beat may be much dislocated, particularly when the sac is large. It is more commonly a dislocation from pressure than from enlargement of the heart itself.

PALPATION.—The area and degree of pulsation are best determined by palpation. When the aneurism is deep seated and not apparent externally, the bimanual method should be used, one hand upon the spine and the other on the sternum. There may be only a diffuse impulse. When the sac has perforated the chest wall the impulse is, as a rule, forcible, slow, heaving, and expansile, and has the same qualities as a forcible apex beat. The resistance may be very great if there are thick laminæ beneath the skin; more rarely the sac is soft and fluctuating. The hand upon the sac, or on the region in which it is in contact with the chest wall, may feel a diastolic shock, often of great intensity, which forms one of the valuable physical signs of aneurism. A systolic thrill is sometimes present, ~~not so often in saccular aneurisms as in the dilatation of the arch~~. The pulsation may sometimes be felt in the suprasternal notch.

PERCUSSION.—The small and deep seated aneurisms are in this respect negative. In the larger tumors, as soon as the sac reaches the chest wall, there is produced an area of abnormal dulness, the position of which depends upon the part of the aorta affected. Aneurisms of the ascending arch grow forward and to the right, producing dulness on one side of the manubrium; those from the transverse arch produce dulness in the middle line, extending toward the left of the sternum, while aneurisms of the descending portion most commonly produce dulness in the left interscapular and scapular regions. The percussion note is flat and gives a feeling of increased resistance.

AUSCULTATION.—Adventitious sounds are not always to be heard. Even in a large sac there may be no murmur. Much depends upon the thickness of the laminæ of fibrin. An important sign, particularly if heard over a dull region, is a ringing, accentuated second sound, a phenomenon rarely missed in large aneurisms of the aortic arch. A systolic murmur may be present; sometimes a double murmur, in which case the diastolic *bruit* is usually due to associated aortic insufficiency. The systolic murmur alone is of little moment in the diagnosis of ~~an~~ aneurism ~~of the~~. A continuous humming top murmur with systolic intensification is heard when the aneurism communicates with the vena cava or the pulmonary artery. With the single stethoscope the shock of the impulse with the first sound ~~is sometimes~~ very marked.

Among OTHER PHYSICAL SIGNS of importance are retardation of the pulse in the arteries beyond the aneurism, or in those involved in the sac. There may, for instance, be a marked difference between the right and left radial, both in volume and time. The blood pressure on the two sides may be unequal. A physical sign of large thoracic aneurism, ~~which I have not seen referred to~~, is obliteration of the pulse in the abdominal aorta and its branches. ~~My~~ attention was called to this in a patient who was stated to have

aortic insufficiency. There was a well-marked diastolic murmur, but in the femorals and in the aorta I was surprised to find no trace of pulsation, and not the slightest throbbing in the abdominal aorta or in the peripheral arteries of the leg. The circulation was, however, unimpaired in them and there was no dilatation of the veins. Attracted by this, I then made a careful examination of the patient's back, when the circumstance was discovered, which neither the patient himself nor any of his physicians had noticed, that he had a very large area of pulsation in the left scapular region. The sac probably was large enough to act as a reservoir annihilating the ventricular systole, and converting the intermittent into a continuous stream.

A remarkable condition suggestive of pneumothorax may be caused by compression of one bronchus by the sac (Newton Pitt). The air is inspired beyond the obstruction, but has difficulty in getting out, so that the lung is gradually distended, causing enlargement of the side with a hyperresonant note on percussion, and on auscultation absence of breath sounds. The X-ray picture may alone decide the diagnosis.

The *tracheal tugging*, a valuable sign in deep-seated aneurisms, was described by Surgeon-Major Oliver, and was specially studied by my colleagues Ross and MacDonnell at the Montreal General Hospital. Oliver gives the following directions: "Place the patient in the erect position, and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb, and use steady and gentle upward pressure on it, when, if dilatation or aneurism exists, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." The tug is sometimes felt more easily if the chin is held down. This is a sign of great value in the diagnosis of deep-seated aneurisms, though it may occasionally be felt in tumors and in the extreme dynamic dilatation of aortic insufficiency. It may be visible in the thyroid cartilage. The trachea may be pushed to one side.

Occasionally a systolic murmur may be heard in the trachea, as pointed out by David Drummond, or even at the patient's mouth, when opened. This is either the sound conveyed from the sac, or is produced by the air as it is driven out of the wind pipe during the systole. Feeble respiration in one lung is a common effect of pressure.

Symptoms.—Broadbent made the useful division of aneurisms of symptoms and aneurisms of physical signs; the former is more commonly seen when the transverse arch is involved, the latter when the ascending portion. There may be no symptoms. A man may present a tumor which has eroded the chest wall without pain or any discomfort. On the other hand, every physical sign may be present without a single symptom.

An important but variable feature in thoracic aneurism is *pain*, which is particularly marked in deep seated tumors. It is usually paroxysmal, sharp, and lancinating, often very severe when the tumor is eroding the vertebræ, or perforating the chest wall. In the latter case after perforation the pain may cease. Anginal attacks are not uncommon, particularly in aneurisms at the root of the aorta. Frequently the pain radiates down the left arm or up the neck, sometimes along the upper intercostal nerves. Superficial tenderness may be felt in the skin over the heart or over the left sternomastoid muscle. *Cough* results either from the direct pressure on the trachea, or is associated

with bronchitis. The expectoration in these instances is abundant, thin, and watery; subsequently it becomes thick and turbid. Paroxysmal cough of a peculiar brazen, ringing character is a characteristic symptom in some cases, particularly when there is pressure on the recurrent laryngeal nerves, or the cough may have a peculiar wheezy quality—the "goose cough."

Dyspnœa, which is common in cases of aneurism of the transverse portion, is not necessarily associated with pressure on the recurrent laryngeal nerves, but may be due directly to compression of the trachea or the left bronchus. It may occur with marked stridor. Loss of voice and hoarseness are consequences of pressure on the recurrent laryngeal, usually the left, inducing either a spasm in the muscles of the left vocal cord or paralysis.

Paralysis of an abductor on one side may be present without any symptoms. It is more particularly, as Semon states, when the paralytic contractures supervene that the attention is called to laryngeal symptoms.

Hæmorrhage in thoracic aneurism may come from (*a*) the soft granulations in the trachea at the point of compression, in which case the sputum is blood tinged, but large quantities of blood are not lost; (*b*) from rupture of the sac into the trachea or a bronchus; (*c*) from perforation into the lung or erosion of the lung tissue. The bleeding may be profuse, rapidly proving fatal, and is a common cause of death. It may persist for weeks or months, in which case it is simply hæmorrhagic weeping through the sac, which is exposed in the trachea. In some instances, even after a very profuse hæmorrhage, the patient recovers and may live for years. A man with well-marked thoracic aneurism, whom I showed to my class at the University of Pennsylvania and who had had several brisk hæmorrhages, died four years after, having in the meantime enjoyed average health. Death from hæmorrhage is relatively more common in aneurism of the third portion of the arch and of the descending aorta.

Difficulty of swallowing is a comparatively rare symptom, and may be due either to spasm or to direct compression. The sound should never be passed in these cases, as the œsophagus may be almost eroded and perforation of the sac has taken place.

Heart Symptoms.—Pain has been referred to; it is often anginal in character, and is most common when the root of the aorta is involved. The heart is hypertrophied in less than one half the cases. The aortic valves are sometimes incompetent, either from disease of the segments or from stretching of the aortic ring.

Among other signs and symptoms, venous compression, which has already been mentioned, may involve one subclavian or the superior vena cava. A curious phenomenon in intrathoracic aneurism is the clubbing of the fingers and incurving of the nails of one hand, of which two examples have been under my care, both without any special distention or signs of venous engorgement. Tumors of the arch may involve the pulmonary artery, producing compression, or in some instances adhesion of the pulmonary segments and insufficiency of the valve; or the sac may rupture into the artery, an accident which happened in two of my cases, producing instantaneous death.

Pupil Symptoms.—These may be due to, first, pressure on the sympathetic, which may cause dilatation of one pupil when the cord is irritated,

contraction when the nerve is paralyzed. Flushing of the side of the face and ear, increased temperature, and sweating may be present. Secondly, as Ainley Walker and Wall have shown, the anisocoria is most frequently due to vascular conditions—with low blood pressure in one carotid the pupil on that side is dilated, with high pressure contracted, and in 26 cases of aneurism they found a relation between the state of the pupil and the arteries on the same side. Thirdly, in a few cases the anisocoria is a parasyphilitic manifestation associated with the Argyll-Robertson phenomenon and absent knee-jerks. The Babinski syndrome.

An X-ray examination should be made in all doubtful cases. The fluoroscope gives an accurate picture of the situation, the size, and the relation to the heart. Even a small sac may be seen. In several cases I have known the diagnosis to rest upon it alone in cases in which scarcely a physical sign was present. Sailer and Pfahler have shown that a condition of tortuosity of the aorta, due to arterio-sclerosis, may exist, suggesting very strongly the presence of aneurism, particularly on examination with the fluoroscope.

The clinical picture of aneurism of the aorta is extremely varied. Many cases present characteristic symptoms and no physical signs, while others have well-marked physical signs and no symptoms.

Diagnosis.—Aneurism of the aorta may be confounded with: (*a*) The violent throbbing impulse of the arch in aortic insufficiency. I have already referred to a case of this kind in which the diagnosis of aneurism was made by several good observers.

(*b*) *Simple Dynamic Pulsation.*—This is common in the abdominal aorta, but is rare in the arch. A case which came under the care of William Murray and Bramwell presented, without any pain or pressure symptoms, pulsation and dulness over the aorta. The condition gradually disappeared and was thought to be neurotic.

(*c*) Dislocation of the heart in curvature of the spine may cause great displacement of the aorta, so that it has been known to pulsate forcibly to the right of the sternum.

(*d*) *Solid Tumors.*—When the tumor projects externally and pulsates the difficulty may be considerable. In tumor the heaving, *expansile* pulsation is absent, and there is not that sense of force and power which is so striking in the throbbing of a perforating aneurism. There is not to be felt, as in aortic aneurism, the shock of the heart sounds, particularly the diastolic shock. Auscultatory sounds are less definite, as large aneurisms may occur without murmurs; and, on the other hand, murmurs may be heard over tumors. The greatest difficulty is in the deep seated thoracic tumors, and here the diagnosis may be impossible. The physical signs may be indefinite. The ringing aortic second sound is of great importance and is rarely, if ever, heard over tumor. Tracheal tugging is here a valuable sign. Pressure phenomena are less common in tumor, whereas pain is more frequent. The general appearance of the patient in aneurism is much better than in tumor, in which there may be cachexia and enlargement of the glands in the axilla or in the neck. Healthy, strong males who have worked hard and have had syphilis are the most common subjects of aneurism. Occasionally cancer of the œsophagus may simulate aneurism, producing pressure on the left bronchus.

(*e*) *Pulsating Pleurisy.*—In cases of *empyema necessitatis*, if the projecting tumor is in the neighborhood of the heart and pulsates, the condition may readily be mistaken for aneurism. The absence of the heaving, firm distention and of the diastolic shock would, together with the history and the existence of pleural effusion, determine the nature of the case. If necessary, puncture may be made with a fine hypodermic needle. In a majority of the cases of pulsating pleurisy the throbbing is diffuse and widespread, moving the whole side.

Prognosis.—The outlook in thoracic aneurism is always grave. Life may be prolonged for some years, but the patients are in constant jeopardy. Spontaneous cure is not very infrequent in the small sacculated tumors of the ascending and thoracic portions. The cavity becomes filled with laminæ of firm fibrin, which become more and more dense and hard, the sac shrinks considerably, and finally lime salts are deposited in the old fibrin. The laminæ of fibrin may be on a level with the lumen of the vessel, causing complete obliteration of the sac. The cases which rupture externally, as a rule, run a rapid course, although to this there are exceptions; the sac may contract, become firm and hard, and the patient may live for five, or even for ten or twenty years. The cases which ~~have~~ lasted longest ~~in my experience have been~~ those in which a saccular aneurism has projected from the ascending arch. One patient in Montreal had been known to have aneurism for eleven years. The aneurism may be enormous, occupying a large area of the chest, and yet life be prolonged for many years. One of the most remarkable instances is the case of dissecting aneurism reported by Graham. The patient was invalided after the Crimean War with aneurism of the aorta, and for years was under the observation of J. H. Richardson, of Toronto, under whose care he died in 1885. The autopsy showed a healed aneurism of the arch, with a dissecting aneurism extending the whole length of the aorta, which formed a double tube.

Treatment.—In a large proportion of the cases this can only be PALLIATIVE. Still in every instance measures should be taken which are known to promote clotting and consolidation within the sac. In any large series of cured aneurisms a considerable majority of the patients have not been known to be subjects of the disease, but the obliterated sac has been found accidentally at the post mortem.

The most satisfactory plan in early cases, when it can be carried out thoroughly, is the modified Valsalva method advised by the late Mr. Tufnell, of Dublin, the essentials of which are rest and a restricted diet. The rest should, as far as possible, be absolute. The reduction of the daily number of heart beats, when a patient is recumbent and without exertion, amounts to many thousands, and is one of the principal advantages of this plan. Mental quiet should also be enjoined. The diet advised by Tufnell is extremely rigid—for breakfast, 2 ounces of bread and butter and 2 ounces of milk or tea; dinner, 3 ounces of mutton and 3 of potatoes or bread and 4 ounces of claret; supper, 2 ounces of bread and butter and 2 ounces of tea. This low diet diminishes the blood volume and is thought also to render the blood more fibrinous. "Total per diem, 10 ounces of solid food and 8 ounces of fluid, and *no more*." This treatment should be pursued for several months, but, except in persons of a good deal of mental stamina, it is impossible to carry

it out for more than a few weeks at a time. It is a form of treatment adapted only to the saccular form of aneurism, and in cases of large sacs communicating with the aorta by a comparatively small orifice the chances of consolidation are fairly good. Unquestionably rest and the restriction of the liquids are the important parts of the treatment, and a greater variety and quantity of food may be allowed with advantage. If this plan can not be thoroughly carried out, the patient should at any rate be advised to live a very quiet life, moving about with deliberation and avoiding all sudden mental or bodily excitement. The bowels should be kept regular, and constipation and straining ~~should be~~ carefully avoided. Of medicines, iodide of potassium, as advised by Balfour, is of great value. It may be given in doses of from 10 to 20 grains (0.6 to 1.3 gm.) three times a day. Larger doses are not necessary. The mode of action is not well understood. It may act by increasing the secretions and so inspissating the blood, by lowering the blood pressure, or ~~as Balfour thinks~~, by causing thickening and contraction of the sac. The most striking effect of the iodide in my experience has been the relief of ~~the~~ pain. The evidence is conclusive that the syphilitic cases are more benefited by it than the non-syphilitic. All these measures have little value unless the sac is of a suitable form and size. The large tumors with wide mouths communicating with the ascending portion of the aorta may be treated on the most approved plans for months without the slightest influence other than reduction in the intensity of the throbbing. A patient with a tumor projecting into the right pleura remained on the most rigid Tufnell treatment for more than one hundred days, during which time he ~~also~~ took iodide of potassium faithfully. The pulsations were greatly reduced and the area of dulness diminished, and we congratulated ourselves that the sac was probably consolidating. Sudden death followed rupture into the pleura, and the sac contained only fluid blood, not a shred of fibrin. In cases in which the tumor is large, or in which there seems to be very little prospect of consolidation, it is perhaps better to advise a man to go on quietly with his occupation, avoiding excitement and worry. Our profession has offered many examples of good work, thoroughly and conscientiously carried out, by men with aneurism of the aorta, who wisely, ~~I think~~, preferred, as did the late Hilton Fagge, to die in harness.

Surgical Measures.—In a few cases consolidation may be promoted in the sac by the introduction of a foreign body, such as wire, ~~horse-hair~~, or by the combination of wiring and electrolysis. Moore, in 1864, first wired a sac, putting in 78 feet of fine wire. Death occurred on the fifth day. Corradi proposed the combined method of wiring with electrolysis, which was first used by Burresi in 1879. His patient lived for three and a half months. Horse-hair, watch-spring wire, catgut, and Florence silk have been used. Hunner reports the statistical results of both methods up to October, 1900. With Moore's method (wiring) 14 cases were treated, 8 of thoracic aneurism, all fatal; 6 aneurisms of the abdominal aorta, 3 of which were successful. Of 23 cases treated by wiring and electrolysis (Moore-Corradi method) 17 were thoracic and 6 abdominal. The thoracic cases of Rosenstirn, Stewart, and Kerr, and the abdominal cases of Noble and Finney (Case V), were successful. In 8 of the 23 cases there were amelioration of symptoms and probable prolongation of life. The most favorable cases are those in which

the aneurism is sacculated, but this is a point not easily determined, and often from a sac particularly favorable for wiring there may be secondary projections of great thinness. The sudden filling by clot of an aneurism of the cœliac axis of the superior mesenteric artery may result fatally from infarct of the intestine.

Other Conditions Requiring Treatment.—Pressure on veins causing engorgement, particularly of the head and arms, is sometimes promptly relieved by free venesection, and, at any time during the course of a thoracic aneurism, if attacks of dyspnœa with lividity supervene, bleeding may be resorted to with great benefit. It has the advantage also of promptly checking the pain, for which symptom, ~~as already mentioned,~~ the iodide of potassium often gives relief. In the final stages morphia is, as a rule, necessary. Dyspnœa, if associated with cyanosis, is best relieved by bleeding. Chloroform inhalations may be necessary. The question sometimes comes up with reference to tracheotomy in these cases of urgent dyspnœa. If it can be shown by laryngoscopic examination that it is due to bilateral abductor paralysis the trachea may be opened, but this is extremely rare, and in nearly every instance the urgent dyspnœa is caused by pressure about the bifurcation. When the sac appears externally and grows large, an ice bag or a belladonna plaster may be applied to allay the pain. In some instances an elastic support may be used with advantage, and I saw a physician with an enormous external aneurism in the right mammary region who for many months had obtained great relief by an elastic support, passing over the shoulder and under the arm of the opposite side.

The calcium salts may be given to influence coagulation, and the nitrites if the blood pressure is high, but rest and diet, restriction of the fluids, and free purgation are usually more effectual than drugs in reducing blood pressure.

ANEURISM OF THE ABDOMINAL AORTA

Of 233 cases collected by Nixon, 207 were in males, 26 in females; 121 were between the ages of twenty-five and forty-five. Nixon reports a case in a syphilitic girl of twenty. Sixteen cases occurred among 16,000 admissions at the Johns Hopkins Hospital.

Pathology.—The sac is most common just below the diaphragm in the neighborhood of the cœliac axis. The tumor may be fusiform or sacculated, and it is sometimes multiple. Projecting backward, it erodes the vertebræ and may cause numbness and tingling in the legs and finally paraplegia, or it may pass into the thorax and burst into the pleura. More commonly the sac is on the anterior wall and projects forward as a definite tumor, which may be either in the middle line or a little to the left. The tumor may project in the epigastric region (which is most common), in the left hypochondrium, in the left flank, or in the lumbar region. When high up beneath the pillar of the diaphragm it may attain considerable size without being very apparent on palpation. When it ruptures into the retro-peritoneal tissues a tumor in the flank may be formed gradually, which enlarges with very little pulsation. It may be mistaken for a rapidly growing sarcoma or for appendicitis, and an operation may be performed.

The **symptoms** are chiefly pain, very often of a neuralgic nature, passing

round to the sides or localized in the back, and more persistent and intense than in any other variety of aneurism. Gastric symptoms, particularly vomiting, may be early and deceptive features. Retardation of the pulse in the femoral artery is a very common symptom.

Diagnosis and Physical Signs.—Inspection may show marked pulsation in the epigastric region, sometimes a definite tumor. A thrill is not uncommon. The pulsation is forcible, expansile, and sometimes double when the sac is large and in contact with the pericardium. On palpation a *definite tumor can be felt.* If large, there is some degree of dulness on percussion, which usually merges with that of the left lobe of the liver. On auscultation, a systolic murmur is, as a rule, audible, and is sometimes best heard at the back. A diastolic murmur is occasionally present, usually very soft in quality. One of the commonest of clinical errors is to mistake a throbbing aorta for an aneurism. It is to be remembered that no pulsation, however forcible, or the presence of a thrill or a systolic murmur, justifies the diagnosis of abdominal aneurism unless there is a *definite tumor which can be grasped and which has an expansile pulsation.* Attention to this rule will save many errors. The throbbing aorta—the "preternatural pulsation in the epigastrium," as Allan Burns calls it—is met with in all neurasthenic conditions, particularly in women. In anæmia, particularly in some instances of traumatic anæmia, the throbbing may be very great. Very frequently a tumor of the pylorus, of the pancreas, or of the left lobe of the liver is lifted with each impulse of the aorta and may be confounded with aneurism. The absence of the forcible expansile impulse and the examination in the knee elbow position, in which the tumor, as a rule, falls forward, and the pulsation is not then communicated, suffice for differentiation. The tumor of abdominal aneurism, though usually fixed, may be very freely movable.

Prognosis.—The outlook in abdominal aneurism is bad. A few cases heal spontaneously. Death may result from (*a*) complete obliteration of the lumen by clots; (*b*) compression paraplegia; (*c*) rupture (which occurred in 152 of the 233 cases in Nixon's series) either into the pleura, retroperitoneal tissues, peritoneum, or the intestines, most commonly into the duodenum; (*d*) embolism of the superior mesenteric artery, producing infarction of the intestines.

The **treatment** is such as already advised in thoracic aneurism. When the aneurism is low down pressure has been successfully applied in a case by Murray, of Newcastle. It must be kept up for many hours under chloroform. The plan is not without risk, as patients have died from bruising and injury of the sac. Nine cases in my series were treated surgically. In two the wiring and electrolysis were followed by great improvement; one man lived for three years.

ANEURISM OF THE BRANCHES OF THE ABDOMINAL AORTA

The **cœliac axis** is itself not infrequently involved in aneurism of the first portion of the abdominal aorta. Of its branches, the **splenic artery** is occasionally the seat of aneurism. This rarely causes a tumor large enough to be felt; sometimes, however, the tumor is of large size. I have reported a case in a man, aged thirty, who had an illness of several months' dura-

tion, severe epigastric pain and vomiting, which led his physicians ~~in New York~~ to diagnose gastric ulcer. There was a deep seated tumor in the left hypochondriac region, the dulness of which merged with that of the spleen. There was no pulsation, but it was thought on one occasion that a *bruit* was heard. The chief symptoms while under observation were vomiting, severe epigastric pain, occasional hæmatemesis, and finally severe hæmorrhage from the bowels. An aneurism of the splenic artery the size of a cocoanut was situated between the stomach above and the transverse colon below, and extended to the right as far as the level of the navel. The sac contained densely laminated fibrin. It had perforated the colon. ~~I have twice seen~~ small aneurisms on the splenic artery. Of 39 instances of aneurism on the branches of the abdominal aorta collected by Lebert, 10 were of the splenic artery.

Of aneurism of the **hepatic artery** Rolland ~~has~~ collected 40 cases (1908), of which 24 were extra-hepatic. In Rolland's case there were three sacs—all intra-hepatic. Rupture took place in 32 cases—in 16 into the peritoneal cavity, in 13 into the bile passages. The sac is rarely large, but in the case of Wollmann's it was as large as a child's head. No case has been diagnosed. Cholelithiasis and duodenal ulcer are the conditions for which it is most likely to be mistaken. In Ross and Osler's case the liver was enlarged, with symptoms of pyæmia.

Aneurism of the **superior mesenteric artery** is not very uncommon. The diagnosis is scarcely possible from aneurism of the ~~arch~~. Plugging of the branches or of the main stem may cause infarction of the bowel.

Renal Artery.—Henry Morris has collected 21 instances of aneurism, 12 of which arose from injury. Many of them were false. Pulsation and a *bruit* are not always present. Four cases were operated upon; three recovered. In a case of Keen's the tumor and the kidney were removed together.

Pulmonary Artery.—Primary aneurism of the trunk is very rare. Of the branches there are two varieties: (*a*) The acute embolic, which may be multiple, arising in connection with thrombi in the veins or a septic endocarditis in the right heart. (*b*) The small aneurisms in the walls of pulmonary cavities, already considered.

ARTERIO-VENOUS ANEURISM

In this form, known to Galen, but first accurately described by the great William Hunter, there is abnormal communication between an artery and a vein. When a tumor lies between the two it is known as *varicose aneurism;* when there is a direct communication without tumor the vein is chiefly distended and the condition is known as *aneurismal varix.*

While it may occur in the aorta, it is much more common in the peripheral arteries as a result of stab or gunshot wounds.

An aneurism of the ascending portion of the arch may open directly into the vena cava. Twenty-nine cases of this lesion have been analyzed by Pepper and Griffith. Cyanosis, œdema, and great distention of the veins of the upper part of the body are the most frequent symptoms, and develop, as a rule, with suddenness. Of the physical signs a thrill is present in some cases. A continuous murmur with systolic intensification is of great diagnostic value. Thurnam (Medico-Chirurgical Transactions, 1840) gave the first accurate

account of this murmur and of this characteristic type of cyanosis. There is only one condition with which it could be confounded, viz., the remarkable cyanosis of the upper part of the body which follows crushing accidents to the thorax. Perforation between the aorta and pulmonary artery causes very much the same symptoms. In a few cases an aneurism of the abdominal aorta perforates the inferior vena cava—œdema and cyanosis of the legs and lower half of the body, and the distinctive thrill and murmur are present.

In the arterio-venous aneurisms which follow stab and bullet wounds of the subclavian, axillary, carotid, femoral, and popliteal arteries the clinical features are most characteristic. First, the veins enlarge as the arterial blood flows under high pressure into them. The affected limb may be greatly swollen and in a young person may lengthen, and the growth of hair is increased. Secondly, a strong thrill is felt, of maximum intensity at the site of the aneurism, but sometimes to be felt at the most distant parts of a limb. Thirdly, the characteristic continuous murmur with systolic intensification is heard. In the external arteries the condition may persist for years before disability is caused by enlargement of the veins and swelling of the limb.

POLYARTERITIS ACUTA NODOSA

(*Periarteritis Nodosa*)

A series of cases has been described in which small aneurisms occur on the arteries of the muscles and viscera. The first case was reported by Kussmaul and Maier, and about 19 cases ~~in all~~ have been described (Dickson). A case, agreeing clinically with the others, ~~has~~ occurred in my wards. No autopsy was permitted, but the nodules were felt in the abdominal wall before death. The case is reported by Sabin (J. H. H. Bulletin, 1901). There are marked thickening of the intima and infiltration of the other coats, with a nuclear growth almost sarcomatous. There are two theories: one, that the nodules are aneurisms due to syphilis or to congenital weakening of the arteries; the other, that they are aneurisms secondary to an inflammatory process like the infectious granulomata.

The cases have occurred chiefly in men between the ages of twenty seven and fifty two; the course is from eight to twelve weeks. The patients complain of weakness. The symptoms correspond with the situation of the lesions; thus their presence in the muscles is associated with pain, weakness, and sometimes paralysis and atrophy. The nodules are abundant in the alimentary tract. The severest symptom is epigastric pain; there is loss of appetite, thirst, vomiting, constipation, or diarrhœa. The disease is febrile at first, but the temperature sinks to subnormal, while the pulse remains rapid. When the cerebral vessels are involved there are headache, excitement, convulsions, and optic neuritis, and the diagnosis of meningitis is made. The anæmia is extreme. In our case the hæmoglobin was 21 per cent., the red blood-cells 1,704,000. The leucocytes reached 116,000, of which 91 per cent. were polymorphonuclear forms. The urine is scanty, of low specific gravity, with albumin and casts. Urea is excreted in small quantities, but the mind is clear.

Lecture on pituitary glandular syndrome

SECTION X

DISEASES OF THE DUCTLESS GLANDS

I. DISEASES OF THE SUPRARENAL BODIES

1. ADDISON'S DISEASE

Definition.—A disease characterized by muscular and vascular asthenia, irritation of the stomach, and pigmentation of the skin; due either to tuberculosis or atrophy of the adrenals, or to degenerative changes in the chromaffin system generally.

The recognition of the disease is due to Addison of Guy's Hospital, whose monograph on "The Constitutional and Local Effects of Disease of the Suprarenal Capsules" was published in 1855.

Etiology.—The disease is rare. Only 17 cases came under my observation in the United States. In large clinics a year or more may pass without a case. Males are more frequently attacked than females. In Greenhow's analysis of 183 cases, 119 were males and 64 females. The majority of cases occur between the twentieth and fortieth years. A congenital case has been described, in which the child lived for eight weeks, and post mortem the adrenals were found to be large and cystic. In a few cases a blow on the abdomen or back has preceded the onset. A certain number of cases have been associated with Pott's disease.

Pathology.—To understand the remarkable character of Addison's disease it is necessary to have an idea of the structure and function of the suprarenal bodies. The cortex of the gland is an epithelial structure, the medulla consists of an irregular meshwork of consecutive tissue, including large multinucleated cells, non-medullated nerve fibres and nerve cells, and in addition large polymorphous cells, which in chromic acid solution take on a brownish pigmentation, and are spoken of as chromaffin cells. Extra-adrenal chromaffin cells are found in the ganglia of the abdominal sympathetic system and in certain structures situated along the course of the aorta, ~~and are~~ known as paraganglia or Zuckerkandl's bodies. These chromaffin cells have also been found in the carotid glands and, according to some authors, in the coccygeal gland, the parovarium and the epidydimis. Collectively these structures are spoken of as the "chromaffin system."

Removal of the suprarenal causes death in animals by progressive weakness and toxæmia.

The specific function of the medullary portion of the gland and of the chromaffin system is to furnish an internal secretion known as epinephrin, which controls blood pressure, acting on peripheral neuro-muscular elements

in the arterioles, promotes the activity of the skeletal muscles, and in some way controls the metabolism of the pigment of the skin. The remarkable discovery of Schäfer and Oliver of the blood pressure raising property of these glands is almost the only positive fact we know in connection with their functions.

Glycosuria is also caused by the injection of epinephrin, and in animals a form of arterio-sclerosis, probably due to the high blood pressure. Following this discovery many theoretical conceptions have been entertained of the relation between a defect of the adrenal secretion and asthenic affections, and it is suggested that adrenal insufficiency itself plays an important rôle in acute infections, in tuberculosis, and many wasting diseases, with which it is interesting to note that increased pigmentation may be associated.

Epinephrin has been shown to be present in the chromaffin bodies, so that we may take it that the chromaffin system has everywhere the special function of providing a material which keeps up the vascular tone.

In some way, too, it controls the pigment metabolism. Abolition of the function of the suprarenals, or marked irritation in the chromaffin tissues in the abdomen, as in tuberculous peritonitis or in aneurism, is associated with a great increase in the pigmentation of the skin.

We have no positive knowledge as to the function of the cortex of the glands. Apparently it has some influence upon sexual activity and pregnancy. This portion of the capsules becomes twice its normal thickness in pregnant rabbits. Hyperplasia of the cortex or tumor formations may be associated with precocious sexual development, and hypoplasia of this part with infantilism. It is ~~also~~ suggested that the cortex produces bodies which neutralize the poisonous products of nitrogenous metabolism and ~~in this way~~ prevent auto-intoxication.

Morbid Anatomy.—There is rarely emaciation or anæmia. Rolleston thus summarizes the condition of the suprarenal bodies in Addison's disease:

"1. The fibro-caseous lesion due to tuberculosis—far the commonest condition found. 2. Simple atrophy. 3. Chronic interstitial inflammation leading to atrophy. 4. Malignant disease invading the capsules, including Addison's case of malignant nodule compressing the suprarenal vein. 5. Blood extravasated into the suprarenal bodies. 6. No lesion of the suprarenal bodies themselves, but pressure or inflammation involving the semilunar ganglia.

"The first is the only common cause of Addison's disease. The others, with the exception of simple atrophy, may be considered as very rare."

The nerve-cells of the semilunar ganglia have been found degenerated and deeply pigmented, and the nerves sclerotic. The ganglia are not uncommonly entangled in the cicatricial tissue about the adrenals. The chromaffin cells in the sympathetic ganglia and in the abdominal plexuses generally disappear. The cases of extensive destruction of the glands without Addison's disease are explained by a persistence of the chromaffin structures elsewhere, while extensive involvement of the extra-capsular chromaffin system may itself be sufficient to cause the symptoms, the adrenals themselves being intact.

Few changes of importance are found in other organs. The spleen is occasionally enlarged; the thymus may be persistent. The other organs show only the alterations associated with a protracted illness.

Symptoms.—In the words of Addison, the characteristic symptoms are "anæmia, general languor or debility, remarkable feebleness of the heart's

action, irritability of the stomach, and a peculiar change of color in the skin."

The onset is, as a rule, insidious. The feelings of weakness, ~~as a rule,~~ usually precede the pigmentation. In other instances the gastro-intestinal symptoms, the weakness, and the pigmentation come on together. There are a few cases in the literature in which the whole process has been acute, following a shock or some special depression. There are three important symptoms:

(*a*) Pigmentation of the Skin.—This, as a rule, first attracts the attention of the patient's friends. The grade of coloration ranges from a light yellow to a deep brown, or even black. In typical cases it is diffuse, but always deeper on the exposed parts and in the regions where the normal pigmentation is more intense, as the areolæ of the nipples and about the genitals; also wherever the skin is compressed or irritated, as by the waistband. At first it may be confined to the face and hands. Occasionally it is absent. Patches showing atrophy of pigment, leucoderma, may occur. The pigmentation is found on the mucous membranes of the mouth, conjunctivæ, and vagina. Pigmentation of the mucous membrane is not distinctive. It has been found in chronic stomach troubles, etc. (Fr. Schultze), and is common in the negro. A patchy pigmentation of the serous membranes has often been found. Over the diffusely pigmented skin there may be little mole like spots of deeper pigmentation, and upon the trunk, particularly on the lower abdomen, it may be "ribbed" like the sand on the seashore.

(*b*) Gastro-intestinal Symptoms.—The disease may set in with attacks of nausea and vomiting, spontaneous in character. Toward the close there may be pain with retraction of the abdomen, and even features suggestive of peritonitis. A marked anorexia may be present. The gastric symptoms are variable throughout the course; occasionally they are absent. Attacks of diarrhœa are frequent and come on without obvious cause.

(*c*) Asthenia, the most characteristic feature of the disease, may be manifested early as a feeling of inability to carry on the ordinary occupation, or the patient may complain constantly of feeling tired. The weakness is specially marked in the muscular and cardio-vascular systems. There may be an extreme degree of muscular prostration in an individual apparently well nourished, whose muscles feel firm and hard. The cardio-vascular asthenia is manifest in a feeble, irregular action of the heart, which may come on in paroxysms, in attacks of vertigo, or of syncope, in one of which the disease may prove fatal. The blood pressure is low, falling to 70 or 80 mm. of Hg. Headache is a frequent symptom; convulsions occasionally occur. Pain in the back may be an early and important symptom.

Anæmia, a symptom specially referred to by Addison, is not common. In a majority of the patients the blood count is normal. McMunn has described an increase in the urinary pigments, and a pigment has been isolated of very much the same character as the melanin of the skin.

The mode of termination is either by syncope, which may occur even early in the disease, by gradual progressive asthenia, or by the development of tuberculous lesions. In ~~two~~ cases ~~I have known~~ a noisy delirium with urgent dyspnœa to precede the fatal event.

Diagnosis.—Pigmentation of the skin is not confined to Addison's disease. The following conditions may give rise to an increase in the pigment; some

of which, *e. g.*, *a* and *b*, are due, as in Addison's disease, to disturbance in the chromaffin system.

(*a*) Abdominal growths—tubercle, cancer, or lymphoma. In tuberculosis of the peritoneum pigmentation is not uncommon.

(*b*) Pregnancy, in which the discoloration is usually limited to the face, the so-called *masque des femmes enceintes.* Uterine disease is a common cause of a patchy melasma.

(*c*) *Hæmochromatosis*, associated with hypertrophic cirrhosis, pigmentation of the skin, and diabetes.

(*d*) In overworked persons of constipated habit and with sluggish livers there may be a patchy staining of the face and forehead.

(*e*) The vagabond's discoloration, caused by the irritation of lice and dirt, which may reach a very high grade, and has sometimes been mistaken for Addison's disease.

(*f*) In rare instances there is deep discoloration of the skin in melanotic cancer, so ~~deep and~~ general that it has been confounded with *melasma suprarenale.*

(*g*) In certain cases of exophthalmic goitre abnormal pigmentation occurs.

(*h*) In a few rare instances the pigmentation in scleroderma may be general and deep.

(*i*) In the face there may be an extraordinary degree of pigmentation due to innumerable small black comedones. If not seen in a very good light, the face may suggest argyria. Pigmentation of an advanced grade may occur in chronic ulcer of the stomach and in dilatation of the organ.

(*j*) Argyria could scarcely be mistaken, and yet ~~I was consulted in a case in which~~ the diagnosis of Addison's disease had been made by ~~several~~ good observers.

(*k*) Arsenic when taken for many months may cause a most intense pigmentation of the skin.

(*l*) With arterio-sclerosis and chronic heart-disease there may be marked melanoderma.

(*m*) In pernicious anæmia the pigmentation may be extreme, most commonly due to the prolonged administration of arsenic.

(*n*) There is a form of deep pigmentation, usually in women, which persists for years without change and without any special impairment of health. ~~I have met with two cases; in one~~ the pigmentation ~~was~~ a little more leaden than is usual in Addison's disease; ~~in both the condition had~~ lasted some ~~years~~.

(*o*) In ochronosis there may be a deep melanotic pigmentation of the face and hands.

In any case of unusual pigmentation these various conditions must be sought for; the diagnosis of Addison's disease is scarcely justifiable without the asthenia. In many instances it is difficult early in the disease to arrive at a definite conclusion. The occurrence of fainting fits, of nausea, and gastric irritability are important indications. As the lesion of the capsules is almost always tuberculous, in doubtful cases the tuberculin test may be used. In ~~two~~ of my cases, robust, healthy men with pigmentation and gastric symptoms, the reaction was obtained.

Prognosis.—The disease is usually fatal. The cases in which the bronzing

Sugar content [illegible] less in same case

Tyrol. Münch. med. Wsch. Nov. 1906. [illegible]

Tolerance for sugar high

in case. Sug. 0.05 %. In 0.1 % in urine

[illegible]

is slight or does not occur run a more rapid course. There are occasionally acute cases which, with great weakness, vomiting, and diarrhœa, prove fatal in a few weeks. In a few cases the disease is much prolonged, even to six or ten years. In rare instances recovery has taken place, and periods of improvement, lasting many months, may occur.

Treatment.—When asthenia appears the patient should be confined to bed and sudden efforts and muscular exercise should not be allowed. Fatal syncope may at any time occur. In three of my cases death was sudden. For the debility arsenic and strychnia are useful; for the diarrhœa large doses of bismuth, and for the irritability of the stomach creosote, hydrocyanic acid, ice, and champagne. The diet should be light and nutritious. As the disease is nearly always tuberculous an open air treatment may be carried out. Tuberculin may be tried, particularly if the case is seen early.

Operation has been suggested. The lesion is usually localized, and nowadays it should not be a difficult matter to remove the diseased glands; but, so far as we know, in animals this is always a fatal procedure, and in any case, unless there were supernumerary adrenals and a considerable portion of the extracapsular chromaffin intact, the operation would be useless.

Adrenal Therapy.—Evidently the relation of Addison's disease to the adrenals is not quite the same as that of myxœdema to the thyroid gland, in which the insufficiency is promptly and permanently relieved by the administration of preparations of the thyroid. The tuberculous nature of the lesions in most of the cases of Addison's disease is in itself an obstacle, and there is usually widespread cicatricial involvement of the sympathetic system. There is now a large series of cases treated with various preparations, but only a very few with satisfactory results. In only two of my patients was there marked improvement. In one, which I have reported, all the severer symptoms disappeared, the pigmentation cleared up, and the patient died subsequently of an acute infection, which apparently had nothing to do with the disease. The adrenals were found sclerotic but not tuberculous. The dried gland may be given in doses of from 5 to 20 grains (0.3 to 1.3 gm.) three times a day. There are also liquid extracts. Epinephrin may also be used and when the blood pressure is low it can be given systematically, carefully testing its effects.

2. OTHER AFFECTIONS OF THE SUPRARENAL GLANDS

Hyperplasia of the cortex has been met with in defective development of the genitals. Enlargement is not uncommon in chronic nephritis and in arterio-sclerosis. The latter has been attributed to the overactivity of the gland. Hypertrophy with tumor has also been associated with a remarkable precocious development of the sexual organs. When one gland is diseased the other may be enlarged. In cases of disease of both glands the chromaffin tissues along the aorta and in the sympathetic system have been found hypertrophied.

Hypoplasia.—There is an interesting association of absence of the adrenals or absence of the medulla with anomalies of the brain, particularly anencephaly. It has been suggested that chronic hypoplasia associated with insufficiency of the internal secretion is responsible for the low blood pressure in the specific fevers and in conditions of debility. Some have attributed the

"white line," the anæmic vaso-motor skin reflex, to adrenal insufficiency, but, in any case, it is too common to be of much import.

Hæmorrhage.—Acute hæmorrhagic adrenalitis presents a picture somewhat resembling acute pancreatitis—a sudden onset with pain, vomiting, profound prostration and death within a few days. In other cases convulsions occur or the patient falls into a typhoid state with profound asthenia. In children the disease may be associated with purpura, both cutaneous and visceral.

Tumors.—Both carcinoma and sarcoma have been described. They are ~~very~~ apt to undergo fatty degeneration and hæmorrhage, so that they may form very large cysts. In children excessive development of the genitals with hair and fat has been found, as noted by Bullock and Sequeira, who ~~have~~ collected a number of cases. On this account a suggestion has been made that the adrenal cortex has an hormonic internal secretion which influences sexual development. Robert Hutchison has described a remarkable syndrome in children of adrenal tumor, exophthalmos, and cranial tumors; and William Pepper (tertius) has described a form characterized by rapid growth, diffuse involvement of the liver, and great distention of the abdomen without ascites or jaundice.

II. DISEASES OF THE THYMUS GLAND

While probably an organ of internal secretion the thymus in structure has little resemblance to the other ductless glands, with the exception of the epiphysis cerebri, and must be classed as an epithelial rather than as a lymphoid organ (Pappenheimer).

At birth the thymus gland weighs about 12 grams; from the first to the fifth year about 23 grams; from the sixth to the tenth year about 26 grams; from the eleventh to the fifteenth year about 37½ grams, and from the sixteenth to the twentieth year about 25½ grams, after which it undergoes a gradual atrophy (Hammar). Involution not taking place, a "persistent thymus" remains.

The function of the gland is not known. Friedleben, whose remarkable monograph (1858) is a storehouse of all knowledge on the subject, found no ill effects from extirpation, but this has not been confirmed. There is an obscure relationship between the thymus and the sexual glands. After castration N. Patton found persistency and hypertrophy of the gland. A disturbance of the normal development of the bones, particularly in ossification, also occurs (Basch) and there is an increase in the excitability of the nerves. The nature of the internal secretion is unknown. Many experiments have been made with extract from the gland, but without definite results.

1. HYPERTROPHY OF THE THYMUS

The size of the gland varies so greatly that it is not easy to define the limits between persistency and enlargement. Between the manubrium sterni and the vertebral column in an infant of eight months the distance is only 2.2 cm. (Jacobi), so that it is easy to understand how an enlarged gland may induce what Warthin calls "thymic tracheostenosis." There would appear to be, as this author suggests, three groups of cases:

adrenals. Stims = the sympathetic system = no effect on an organ if no sympath. stim. acts as dilator solution.

emotion causes adrenalin discharge. asphyxia does the same.

effect = dig. syst. quiet & blood shifted to active muscles nerves & resp organs
discharge of dextrose, hyperglycaemia muscles stimulated
bronchioles dilated. up rate & force of beat & resp. of blood. reduces but gen.

so effect of pres on adrenal stim. in fear etc. to detriment of vegetative activity
note in high b.p. cases. diagnosis

injection of adr = gen. stim. of sympathetic system = so prob. maintains sympathetic tone

Injection of adrenalin much best in high b.p.

Injection terms [illegible] is doubtful = it may be that adr. exp. result in vaso constriction for maintenance of sympath. irritability

note - no test for sympathicotonia by epinephrin in graves dis.
Epin has little influence on sympathetic system proper. An exaggerated reaction to epin. does not indicate sympath. instability but sympath. dep. (Pilocarpin would be useful)
Dilatation of adrenals. low b.p. due to weakness of heart - prob. no vessels)
Prob. a person with adr. dilatation is due to loss of cortical tissue.

Hypertrophy of Thymus. Compresses trachea, jugulars and subclavian veins from brain & arm. This impedes circ. in thymus & vicious circle. Death imminent if thymus Bronchial mucous membrane may also be congested, pulm. veins congested. Severe asphyxia may develop.
Sudden paralysis may attend hyp. of thymus
Symptoms in adolescence also marked — especially with hypoplasia.
the endocrine system [illegible]

Friedleben. Thymus Emerson. Arch. Int. Med
1914. XIII. p. 169

Men: Scantiness of hair on face (chin, upper lip)
" " axillae, chest.
" " pubic or feminine distrib

Slender thorax.
rounded arms & thighs — & arching of clavicles.
hypoplastic external genitals &
perhaps cryptorchism
Delicate velvety skin.

hypoplasia of lymphatic tissue of throat.
increase in palp. cervical & axillary glands

Women. Skin:
Scanty axillary, pubic & perineal hair
hypoplasia of genitals.
Slender thorax & extremities.

Thymus may not be enlarged.

(*a*) Thymic stridor, either congenital or developing soon after birth, varying in intensity and aggravated by crying and coughing.

(*b*) Thymic asthma, sometimes known as Copp's or Miller's asthma, which is an exaggerated and more persistent form of the stridor. While much dispute exists as to this form, there can be no doubt as to its occurrence, as there are cases, those reported by Siegel and König, for example, in which complete relief has followed removal of the gland. Olivier has collected 39 cases of thymectomy with 24 recoveries.

(c) Lastly, in a few cases sudden death has been met with, usually in connection with the condition of lymphatism about to be described.

Persistence of the gland has been met with in many affections, such as Graves' disease, Addison's disease, acromegaly, myasthenia gravis, rickets, etc. Many observers have regarded the association of an enlargement with Graves' disease as more than accidental and as a sort of compensatory process.

2. ATROPHY OF THE THYMUS

This is met with accidentally in children who show no special pathological changes, especially as Ruhräh has shown, in marasmus and the chronic wasting disorders of children.

Of other morbid conditions met with, hæmorrhages are not uncommon. Many mediastinal tumors originate in the remnants of the thymus; dermoid tumors and cysts have also been met with; tuberculosis and syphilis of the gland are occasionally seen.

The condition described by Dubois, met with sometimes in the subjects of congenital syphilis, in which there are fissure like cavities in the gland filled with a purulent fluid, is stated by Chiari and by Dudgeon to be a post mortem softening.

3. STATUS THYMICO-LYMPHATICUS

(*Lymphatism*)

Definition.—A condition in children of hyperplasia of the lymphatic tissues and of the thymus, in association with a flabby, fat overgrowth of the body and hypoplasia of the heart and blood vessels.

The subjects of lymphatism, as this condition has been called, have a lowered resistance, and are liable to sudden death from trifling and apparently inadequate causes. The so-called "thymic death" has been much discussed. In young infants who have been found dead in bed, or who have been suddenly attacked with dyspnœa and cyanosis and died in a few minutes, the thymus gland has been found enlarged. Paltauf increased our knowledge of this condition when he described (in 1889) a *lymphatic constitution*, in which there was hyperplasia of the general lymphatic apparatus, enlargement of the thymus and of the spleen, with a fat, flabby state of the body.

Pathology.—The children have a well-developed panniculus adiposus, are often large for their age, and with relatively large heads. There are hyperplasia of the lymphatic apparatus and structures of the tonsillar ring, moderate enlargement of the external and internal lymph glands, and hyperplasia of the solitary and agminated follicles of the small and large intestines. In the mod-

erately enlarged spleen the Malphigian bodies may stand out very prominently, and if anæmic look like large tubercles. The thymus may measure as much as 10 cm. in length, is swollen and soft, and on section exudes a milky white fluid. The bone marrow has been found hyperplastic, and in young adults the yellow is replaced by the red marrow. A small heart, small aorta and peripheral vessels have been found, and sometimes an associated condition of rickets.

Symptoms.—The subjects of lymphatism are usually fat, often anæmic and flabby, but are regarded as in good health. It is usually met with in children, but the condition may persist beyond puberty, and is seen in the flabby young adults with pasty complexions, large heads, and slender skeletons.

What has called special attention to the condition is the tragedy of sudden death following trifling causes—as the prick of a hypodermic needle, a sudden plunge into cold water, but much more often in anæsthesia, either by ether or chloroform, when an amount has been given, not itself lethal. Possibly, too, some of the sudden deaths of convalescents from infectious fevers are from this cause, and the remarkable cases of sudden death during bathing, or of persons who have fallen into the water, and though immediately rescued were dead. Two explanations have been offered of this sudden death: first, that it is due to mechanical pressure of the enlarged thymus on the trachea. This is not likely, as the majority of these cases have shown no previous signs of thymic asthma, and in only one of Blumer's 9 cases was there any evidence of this. The other view is that it is a toxæmia from an overproduction of the internal secretion of the thymus and of the lymph glands.

Diagnosis.—Suspected cases should be carefully examined before trifling operations. The enlargement of the superficial glands of the tonsillar tissues and of the spleen is easily determined. A persistence of the thymus is indicated by an area of dulness at the upper part, and particularly to the left of the sternum merging with the cardiac flatness and which shifts upward with extreme retraction of the head and neck (Boggs). A distinct bulging may be seen, or the top of the gland may be felt in the episternal notch. The X-ray picture may show a definite shadow. With the laryngoscope a narrowing of the windpipe may be seen. A careful blood count should always be made, as there may be a marked lymphocytosis. It is not easy to determine the existence of vascular hypoplasia.

Treatment.—Lymphatism is a condition out of which the child gradually grows. A general tonic treatment with iron and arsenic should be given. A large thymus causing compression should be removed. ~~A. Friedlander has treated cases~~ successfully with the X-ray.

III. DISEASES OF THE THYROID GLAND

1. CONGESTION

At puberty, in girls, often at the onset of menstruation, the gland enlarges; in certain women the neck becomes fuller at each menstruation, and it was an old idea that the gland enlarged at or after defloration. The slight enlargement at puberty may persist for months and cause uneasiness, but, as a rule, it disappears completely. ~~I~~ do not remember a single instance in which the

Many cases have been

Slight enlarg. [illegible]

Tub. [illegible] in Diag. as [illegible]

goitre has remained, though, of course, such a possibility has to be considered. From mechanical causes, as tight collars, repeated crying, or prolonged use of the voice, the gland may swell for a short time.

2. THYROIDITIS

Etiology.—Inflammation of the gland, which is nearly always secondary to some infection, may be simple or purulent. It has been met with most frequently in typhoid fever, small-pox, measles, pneumonia, rheumatic fever, and mumps. Epidemics of thyroiditis have been reported. It is a rare disease in ordinary hospital practice, and did not occur in one of our series of 1,500 cases of typhoid fever.

Symptoms.—The whole gland may be involved, or only one lobe. There are swelling, pain on pressure, redness over the affected part, and, when suppuration occurs, softening or fluctuation. Often the acute inflammation subsides spontaneously. Myxœdema has followed destruction of the entire gland by acute suppuration.

A remarkable sclerotic thyroiditis has been described by Riedel and is sometimes called after his name. It is important, as, in the rapidity of its evolution and in the production of a diffuse tumor involving the whole gland, the clinical picture may resemble cancer. The gland becomes firmly fixed to the surrounding parts and serious effects may be produced by compression of the trachea and of the recurrent laryngeal nerves. The cut section of the gland is white and smooth, and ~~microscopically~~ consists of a dense fibrous tissue.

3. TUMORS OF THE THYROID

Of these the most important are:

(*a*) Infective granulomata—tuberculosis, actinomycosis, and syphilis. Cases are very rarely ~~met with~~. Swelling of the gland has been seen in ~~the~~ recent syphilitic infection, and gummata may occur in the congenital form.

(*b*) Adenomata, simple or malignant. The latter may cause extensive metastases, as in the case reported by Hayward, in which tumors resembling thyroid tissue occurred in the lungs and various bones.

(*c*) Cancer and sarcoma, which are rare, have a surgical interest.

4. ABERRANT AND ACCESSORY THYROIDS

In various places, from the root of the tongue to the arch of the aorta, fragments of thyroidal tissue have been found. These aberrant portions of the gland are very apt to enlarge and undergo cystic degeneration. In the mediastinum they may form large tumors, and in the pleura ~~I have seen~~ an accessory cystic thyroid occupy the upper portion, and a case was reported by F. A. Packard, in which the cystic gland filled nearly the entire side. The so-called lingual thyroid is not uncommon, varying in size from a hemp seed to a pea, usually free in the deep muscles of the tongue, or attached to the hyoid bone. When enlarged the lingual goitre may form a tumor of considerable size. The true thyroid gland has been absent, and removal of the lingual goitre has been followed by myxœdema.

5. GOITRE

(*Struma, Bronchocele*)

Definition.—A chronic enlargement of the thyroid gland, of unknown origin, occurring sporadically or endemically.

Distribution.—Goitre, ~~on the whole, is rare~~ in the United States, it is perhaps most common in the region of the Great Lakes. In an investigation in Michigan, Dock found a large number of cases and the disease is not very uncommon in Lower Canada. In England it is common in certain regions; the Thames valley, the Dales, Derbyshire, Sussex, and Hampshire. It is very prevalent about Oxford and the upper Thames valley. In Switzerland, in the mountains of Germany and Austria, the mountainous districts of France, and in the Pyrenees the disease is very prevalent. In regions of Central Asia, in the Abyssinian mountains, and in the Himalayas there are many foci of the disease.

Etiology.—The disease is rarely congenital except in very goitrous districts. Cases are most common at or about puberty, and the tendency diminishes after the twentieth year. Women are much more frequently attacked than men, in a proportion of 6 or 8 to 1.

In its endemic form the disease occurs at every latitude and in every altitude, in valleys and in plains, and in various climates. It seems to be much less prevalent by the seashore.

The cause is obscure. The water in goitrous districts is hard, rich in lime and magnesia, poor in iodine, and (so Rédin affirms of the Swiss waters) with a high degree of radio-activity. Others speak of a "miasma" of the soil which gets into the drinking water. McCarrison in Kashmir found that the specific agent could be killed by boiling the water and that it did not pass a Berkefeld filter. He produced goitre in himself and in others by the daily consumption of the residue of the filter, but the residue when boiled was harmless. The disease was transmitted to goats who drank water contaminated by goitre patients. There are "goitre springs" and "goitre wells." These and other facts strongly suggest a specific organism; and this view is supported by the remarkable outbreaks of acute goitre in schools, lasting for a few months and disappearing. In one such outbreak 161 boys among 350 and 245 girls among 381 were attacked (Guillaume).

Morbid Anatomy.—Usually the whole gland is involved, but one lobe only may be attacked. When the enlargement is uniform, and the appearance of the gland natural, it is spoken of as parenchymatous goitre; when the blood vessels are very large, vascular goitre. In both forms there is an increase in the colloid material of the follicles. Degenerations of various kinds are common, particularly cystic, in which there are many large and small cavities with colloid contents. In some of these cystic forms there are papillary ingrowths into the alveoli. Sometimes the cysts contain blood and extensive hæmorrhages occur in the gland.

Symptoms.—When small a goitre is not inconvenient, but when large pressure symptoms may cause the patient to seek relief. The windpipe may be flattened from pressure, usually of an enlarged isthmus, or it is narrowed by circular compression. The symptoms are more or less marked stridor and

Annalen

1). Exopth. Gott

2). [illegible]

3). Cornutia [illegible]

[illegible]

cough, which may persist for years without special aggravation. They may be present with very large glands, or with the small encircling goitre, or with the goitre which passes deeply beneath the sternum. Pressure on the recurrent nerves may cause attacks of dyspnœa, particularly at night, and the voice may be altered. Pressure on the vagus is not common. Sometimes there is difficulty in swallowing, and the veins of the neck may be compressed. The heart is often involved, either from pressure on the vagi, or there is dilatation of the organ associated with dyspnœa. This is sometimes spoken of as the "goitre heart" in contra-distinction to the cardiac condition in Graves' disease.

Prognosis.—Many cases in the young get well; too often in goitrous districts the tumor persists. It may disappear on leaving the district. Many cases get well without medical treatment, but when pressure symptoms occur surgery gives relief.

Treatment.—In goitrous districts the drinking water should be boiled. Iodine in some form is used extensively, and often is curative. Its effect is to stimulate the gland to healthy action. In young people 2 to 5 grains (0.13 to 0.3 gm.) of potassium or sodium iodide may be given daily. Iodine injections into the gland have been used but are not advisable. Iodine may be applied externally as an ointment (5 per cent.). The X-rays have been tried with success. When the gland is large, surgical measures must be resorted to.

6. HYPOTHYROIDISM

(*Cretinism and Myxœdema*)

Definition.—A constitutional affection due to the loss of function of the thyroid gland, characterized clinically by a myxœdematous condition of the subcutaneous tissues and mental failure, and anatomically by atrophy of the thyroid gland.

History.—As early as 1859 Schiff had noted that in the dog removal of the gland was followed by certain symptoms. Gull described "A cretinoid change in women," and in the eighties the observations of Ord and other English physicians separated a well defined clinical entity called "myxœdema."

Kocher (in 1883) reported that 30 of his first 100 thyroidectomies had been followed by a very characteristic picture, to which he gave the name "cachexia strumipriva," an observation which had already been made in the previous year by the Reverdins, who also had recognized the relation of this change to the disease known as "myxœdema." The researches of Horsley, and the elaborate investigation of the Committee of the Clinical Society of London, made it clear that the changes following complete removal of the gland, the so-called cachexia strumipriva, myxœdema, and the sporadic cretinism, were one and the same disease, due to the loss of the function of the thyroid gland. Schiff and Horsley demonstrated that animals could be saved by the transplantation of the glands. Lastly came the discovery of George Murray and Howitz that feeding with the thyroid extract replaced the gland function, and cured the disease. The activity of the gland is in some way connected with the metabolism of iodine. Baumann determined the presence of this body in the secretion of the thyroid as an organic combination which

he called iodothyrin, not to be confounded with the simple extract of the gland, which is usually called thyreoidin.

The outcome of a host of researches has been the recognition of the enormous importance of the internal secretion of the gland, which is essential for the normal growth of the body in childhood, and for the maintenance of the proper metabolism of the epidermic tissues and of the brain.

Clinical Forms.—There are three groups of cases—cretinism, myxœdema proper, and operative myxœdema. To Felix Simon is due the credit of recognizing that these were one and the same condition and all due to loss of function of the thyroid gland.

Cretinism.—Two forms are recognized—the sporadic and the endemic. In the *sporadic* form the gland may be congenitally absent, or is atrophied after one of the specific fevers, or the condition develops with goitre. The disease is not very uncommon. I was able to collect the histories of 58 cases in a few years in the United States and Canada. It is more common in females than in males—35 in my series.

Morbid Anatomy.—Absence of the gland, or complete fibrous atrophy, is the common condition. Goitre with any trace of gland tissue is rare. In the sporadic form sometimes the hypophysis and thymus have been found enlarged. Arrest of development, a brachycephalic skull in the endemic, and a doliocephalic in the sporadic form, are the chief skeletal changes.

Symptoms.—In the congenital cases the condition is rarely recognized before the infant is six or seven months old. Then it is noticed that the child does not grow so rapidly and is not bright mentally. The tongue looks large and hangs out of the mouth. The hair may be thin and the skin very dry. Usually by the end of the first year and during the second year the signs become very marked. The face is large, looks bloated, the eyelids are puffy and swollen; the alæ nasi are thick, the nose looks depressed and flat. Dentition is delayed, and the teeth which appear decay early. The abdomen is swollen, the legs are thick and short, and the hands and feet are undeveloped and pudgy. The face is pale and sometimes has a waxy, sallow tint. The fontanelles remain open; there is much muscular weakness, and the child can not support itself. In the supraclavicular regions there are large pads of fat. The child does not develop mentally and may lapse into a state of imbecility.

In cases in which the atrophy of the gland follows a fever the condition may not come on until the fourth or fifth year, or later. This is really, as Parker determined, a juvenile myxœdema. In a few of the sporadic forms cretinism develops with an existing goitre. It may retard development, bodily and mental, without ever progressing to complete imbecility.

Endemic Cretinism.—This occurs wherever goitre is very prevalent, as in parts of Switzerland, Savoy, Tyrol, and the Pyrenees. It formerly prevailed in parts of England. I know of no centres in the United States or Canada. The clinical features of the disease are the same as in the sporadic form, stunted growth and feeble mind, plus goitre. To some poison in the water—mineral or organic—the thyroid changes have been attributed, but whatever the toxic agent may be, it is the interference with the function of the gland that leads to the cretinous change in the body.

The *diagnosis* is very easy after one has seen a case, or good illustrations.

Oculo-cardiac reflex marked.
Hypothyroidism leaves sympathetic without nat. stim. from thyroid.
So opposition seems a vagotony result.
Thyroid less exophthalmic..

Infants a year or so old sometimes become flabby, lose their vivacity, or show a protuberant abdomen, and lax skin with slight cretinoid appearance. These milder forms, as they have been termed, are probably due to transient functional disturbance in the gland.

Myxœdema of Adults (*Gull's Disease*).—Women are much more frequently affected than men—in a ratio of 6 to 1. The disease may affect several members of a family, and it may be transmitted through the mother. In some instances there has been first the appearance of exophthalmic goitre. Though ~~occurring~~ most commonly in women, it seems to have no special relation to the catamenia or to pregnancy; the symptoms of myxœdema may disappear during pregnancy or may develop post partum. Myxœdema and exophthalmic goitre may occur in sisters. It is not so common in America as in England. In sixteen years I saw only 10 cases in Baltimore, 7 of which were in the hospital. C. P. Howard ~~has~~ collected 100 American cases, of which 86 were in women. The symptoms of this form, as given by Ord, are marked increase in the general bulk of the body, a firm, inelastic swelling of the skin, which does not pit on pressure; dryness and roughness, which tend with the swelling to obliterate in the face the lines of expression; imperfect nutrition of the hair; local tumefaction of the skin and subcutaneous tissues, particularly in the supraclavicular region. The physiognomy is altered in a remarkable way: the features are coarse and broad, the lips thick, the nostrils broad and thick, and the mouth is enlarged. Over the cheeks, sometimes the nose, there is a reddish patch. There is a striking slowness of thought and of movement. The memory becomes defective, the patients grow irritable and suspicious, and there may be headache. In some instances there are delusions and hallucinations, leading to a final condition of dementia. The gait is heavy and slow. The temperature may be below normal. The functions of the heart, lungs, and abdominal organs are normal. Hæmorrhage sometimes occurs. Albuminuria is sometimes present, more rarely glycosuria. Death is usually due to some intercurrent disease, most frequently tuberculosis (Greenfield). The thyroid gland is diminished in size and may become completely atrophied and converted into a fibrous mass. The subcutaneous fat is abundant, and in one or two instances a great increase in the mucin has been found. The larynx is also involved.

The course of the disease is slow but progressive, and extends over ten or fifteen years. A condition of acute and temporary myxœdema may develop in connection with enlargement of the thyroid in young persons. Myxœdema may follow exophthalmic goitre. In other instances the symptoms of the two diseases have been combined. ~~I have reported a~~ case ~~in which~~ a young man became bloated and increased in weight enormously during three months, then had tachycardia with tremor and active delirium, and died within six months of the onset of the symptoms.

Operative Myxœdema; Cachexia Strumipriva.—Horsley showed that complete removal of the thyroid in monkeys was followed by the production of a condition similar to that of myxœdema and sometimes associated with spasms or tetanoid contractures, and followed by apathy and coma. An identical condition sometimes follows extirpation of the thyroid in man. The disease follows only a certain number of total and a much smaller proportion of partial removals of the thyroid gland. Of 408 cases, in 69 the operative

myxœdema occurred (Kocher). If a small fragment of the thyroid remains, or if there are accessory glands, which in animals are very common, the symptoms do not develop. Operative myxœdema is very rare in America. A few years ago I was able to find only two cases, one of which, McGraw's, referred to in previous editions of this work, has since been cured.

The *diagnosis* of myxœdema is easy, as a rule. The general aspect of the patient—the subcutaneous swelling and the pallor—suggests Bright's disease, which may be strengthened by the discovery of tube casts and of albumin in the urine; but the solid character of the swelling, the exceeding dryness of the skin, the yellowish white color, the low temperature, the loss of hair, and the dull, listless mental state should suffice to differentiate the two conditions. In dubious cases not too much stress should be laid upon the supraclavicular swellings. There may be marked fibro-fatty enlargements in this situation in healthy persons, the supraclavicular pseudo-lipomata of Verneuil.

Treatment.—The patients suffer in cold and improve greatly in warm weather. They should therefore be kept at an even temperature, and should, if possible, move to a warm climate during the winter months. Repeated warm baths with shampooing are useful. Our art has made no more brilliant advance than in the cure of these disorders due to disturbed function of the thyroid gland. That we can to-day rescue children otherwise doomed to helpless idiocy—that we can restore to life the hopeless victims of myxœdema—is a triumph of experimental medicine for which we are indebted very largely to Victor Horsley and to his pupil Murray. Transplantation of the gland was first tried; then Murray used an extract subcutaneously. Hector Mackenzie in London and Howitz in Copenhagen introduced the method of feeding. We now know that the gland, taken either fresh, or as the watery or glycerin extract, or dried and powdered, is equally efficacious in a majority of all the cases of myxœdema in infants or adults. Many preparations are now on the market, but it makes little difference how the gland is administered. The dried powdered gland and the glycerin extract are most convenient. It is well to begin with the powdered gland, 1 grain (0.065 gm.) three times a day, of the Parke Davis preparation, or one of the Burroughs and Welcome tablets. The dose may be increased gradually until the patient takes 10 or 15 grains (0.6 gm. to 1 gm.) in the day. In many cases there are no unpleasant symptoms; in others there are irritation of the skin, restlessness, rapid pulse, and delirium; in rare instances tonic spasms, the condition to which the term *thyroidism* is applied. The results, as a rule, are most astounding—unparalleled by anything in the whole range of curative measures. Within six weeks a poor, feeble-minded, toad-like caricature of humanity may be restored to mental and bodily health. Loss of weight is one of the first and most striking effects; one of my patients lost over 30 pounds within six weeks. The skin becomes moist, the urine is increased, the perspiration returns, the temperature rises, the pulse rate quickens, and the mental torpor lessens. Ill effects are rare. Two or three cases with old heart lesions have died during or after the treatment; in one instance a temporary condition of Graves' disease was induced.

The treatment, as Murray suggests, must be carried out in two stages—one, early, in which full doses are given until the cure is effected; the other, the permanent use of small doses sufficient to preserve the normal metabolism.

Kendall. J. A. M. A. 1915. 64. 2042.

Thyroid proteins. acid insoluble A
" soluble B.

from A. crystalline compound. 6:90 I. - produces
tumor in pulse rate, metabolism & nervous milieu
in excess toxic symptoms

In Exophth. goitre - Secretory capacity increased
" decreased
storage

B. no toxic effects. but in hyperthyroidism
shows physiological activity

Gen. metabolism shows an increase - rest decreases. (Mean rate)
increase basal metabolism.

In the cases of cretinism it seems to be necessary to keep up the treatment indefinitely. I have seen several instances of remarkable relapse follow the cessation of the use of the extract.

7. HYPERTHYROIDISM; EXOPHTHALMIC GOITRE

(*Graves', Basedow's, or Parry's Disease*)

Definition.—A disease characterized by goitre, exophthalmos, tachycardia, and tremor, associated with a perverted or hyperactive state of the thyroid gland.

Historical Note.—In the posthumous writings of Caleb Hillier Parry (1825) is a description of 8 cases of Enlargement of the Thyroid Gland in Connection with Enlargement or Palpitation of the Heart. In the first case, seen in 1786, he also described the exophthalmos: "The eyes were protruded from their sockets, and the countenance exhibited an appearance of agitation and distress, especially in any muscular movement." The Italians claim that Flajani described the disease in 1800. I have not been able to see his original account, but Moebius states that it is meagre and inaccurate, and bears no comparison with that of Parry. If the name of any physician is to be associated with the disease, undoubtedly it should be that of the distinguished old Bath physician. Graves described the disease in 1835 and Basedow in 1840.

Etiology.—*Age*—In Sattler's collection of 3,477 cases only 184 were under the age of sixteen.

Sex—In England and America the proportion of females is greatly in excess, as much probably as 20 to 1, but in Sattler's collected cases from the literature the ratio was 5.4 to 1, which would indicate marked differences in different countries.

A strong *family predisposition* may exist and five or six members may be affected. *Fright* is a rare cause. Various depressive influences, such as worry, nervous strain, disappointment in love, illnesses, and mental shocks, as well as dread of the disease itself, may have an important influence.

Pathology.—The disease is regarded by some as a pure neurosis, in favor of which are urged the onset after a profound emotion, the absence of lesions, and the cure which has followed in a few cases after operations upon the nose. Of late the views of Moebius and Greenfield have prevailed, that exophthalmic goitre is primarily a disease of the thyroid gland (*hyperthyroidism*), in antithesis to myxœdema (*athyroidism*). The clinical contrast between these two diseases is most suggestive—the increased excitability of the nervous system, the flushed, moist skin, the vascular erythism in the one; the dull apathy, the low temperature, slow pulse, and dry skin of the other. The changes in the gland in exophthalmic goitre are, as shown by Greenfield, those of an organ in active evolution—viz., increased proliferation, with the production of newly formed tubular spaces and absorption of the colloid material which is replaced by a more mucinous fluid. The thyroid extract given in excess produces symptoms not unlike those of Parry's disease—tachycardia, tremor, headache, sweating, and prostration. Beclère has reported a case in which exophthalmos developed after an overdose. Use of the thyroid extract usually aggravates the symptoms of exophthalmic goitre. The most successful line of treatment

has been that directed to diminish the bulk of the goitre. These are some of the considerations which favor the view that the symptoms are due to disturbed function of the thyroid gland, probably to hypersecretion of materials which induce a sort of chronic intoxication. Myxœdema may develop in the late stages, and there are transient œdema and in a few cases scleroderma, which indicate that the nutrition of the skin is involved.

Anatomical Changes.—In rare instances the thyroid gland has been stated to be normal. In the majority of cases there is active hyperplasia of the gland, with enlarged and newly formed follicles, and an increase in the lymphoid tissue of the gland stroma. Involuntary and regressive changes are common; the hyperplasia may cease and the gland returns to the colloid state. Finally, in certain cases, atrophy of the cell elements takes place.

The iodine content of the gland bears a direct relationship to the amount of colloid; the gland in hyperplasia has the lowest percentage, the pure colloid glands the highest.

Symptoms.—Acute and chronic forms may be recognized. In the acute form the disease may arise with great rapidity. In a patient of J. H. Lloyd's, of Philadelphia, a woman, aged thirty-nine, who had been considered perfectly healthy, but whose friends had noticed that for some time her eyes looked rather large, was suddenly seized with intense vomiting and diarrhœa, rapid action of the heart, and great throbbing of the arteries. The eyes were prominent and staring and the thyroid gland was found much enlarged and soft. The gastro-intestinal symptoms continued, the pulse became more rapid, the vomiting was incessant, and the patient died on the third day of the illness. Only the abdominal and thoracic organs could be examined and no changes were found. I saw two rapidly fatal cases at the Philadelphia Hospital, one of which, under F. P. Henry's care, had marked cerebral symptoms. The acute cases are not always associated with delirium.

More frequently the onset is gradual and the disease is chronic. There are four characteristic symptoms of the disease—tachycardia, exophthalmos, enlargement of the thyroid, and tremor.

Tachycardia.—Rapid heart action is only one of a series of remarkable vascular phenomena in the disease. The pulse rate at first may be not more than 95 or 100, but when the disease is established it may be from 140 to 160, or even higher. Irregularity is not common, except toward the close. In a well developed case the visible area of cardiac pulsation is much increased, the action is heaving and forcible, and the shock of the heart sounds is well felt. The large arteries at the root of the neck throb forcibly. There is visible pulsation in the peripheral arteries. The capillary pulse is readily seen, and there are few diseases in which one may see at times with greater distinctness the venous pulse in the veins of the hand. The throbbing pulsation of the arteries may be felt even in the finger tips. Vascular erythema is common—the face and neck are flushed and there may be a widespread erythema of the body and limbs. On auscultation murmurs are usually heard over the heart, a loud apex systolic and loud bruits at the base and over the manubrium. The sounds of the heart may be very intense. In rare instances they may be heard at some distance from the patient; according to Graves, as far as four feet. Attacks of acute dilatation of the heart may occur with dyspnœa, cough, and a frothy bloody expectoration.

EXOPHTHALMOS.—A characteristic facial aspect is given to Graves' disease by the staring expression, caused in part by protrusion of the eyeballs, but more particularly by retraction of the lids exposing the scleræ above and below the corneæ. The exophthalmos, which may be unilateral, usually follows the vascular disturbance. The protrusion may become very great and the eye may even be dislocated from the socket, or both eyes may be destroyed by panophthalmitis, a condition present in one of Basedow's cases. The vision is normal. Graefe noted that when the eyeball is moved downward the upper lid does not follow it as in health. This is known as Graefe's sign. The palpebral aperture is wider than in health, owing to spasm or retraction of the upper lid (Stellwag's sign). The patient winks less frequently than in health. There is marked tremor of the lids and they contract spasmodically in advance of the elevating eyeball. Moebius has called attention to the lack of convergence of the two eyes. Changes in the pupils and in the optic nerves are rare. Pulsation of the retinal arteries is common.

ENLARGEMENT OF THE THYROID is the rule. It may be general or in only one lobe, and is rarely so large as in ordinary goitre. It may be absent. The swelling is firm, but elastic. There are rarely pressure signs. The vessels are usually much dilated, and the whole gland may be seen to pulsate. A thrill may be felt on palpation and on auscultation a loud systolic murmur, or more commonly a *bruit de diable*. A double murmur is common and is pathognomonic (Guttmann).

TREMOR is the fourth cardinal symptom, and was really first described by Basedow. It is involuntary, fine, about eight to the second. It is of great importance in the diagnosis of the early cases.

Other symptoms are anæmia, emaciation, and slight fever. Attacks of vomiting and diarrhœa may occur. The latter may be very severe and distressing, recurring at intervals. The greatest complaint is of the forcible throbbing in the arteries, often accompanied with unpleasant flushes of heat and profuse perspirations. An erythematous flushing is common. Pruritus may be a severe and persistent symptom. Multiple telangiectases have been described. Solid, infiltrated œdema is not uncommon. It may be transitory. A remarkable myxœdematous state may supervene. Pigmentary changes are very common. They may be patchy or generalized. The coexistence of scleroderma and Graves' disease has been frequently noticed. Irritability of temper, change in disposition, and great mental depression have been described. An important complication is acute mania, in which the patient may die in a few days. Weakness of the muscles is not uncommon, particularly a feeling of "giving way" of the legs. If the patient holds the head down and is asked to look up without raising the head, the forehead remains smooth and is not wrinkled, as in a normal individual (Joffroy). A feature of interest noted by Charcot is the great diminution in the electrical resistance, which may be due to the saturation of the skin with moisture owing to the vaso-motor dilatation (Hirt). Bryson has noted the fact that the chest expansion may be greatly diminished. The emaciation may be extreme. Glycosuria and albuminuria are not infrequent complications. True diabetes and may occur.

The course of the disease is usually chronic, lasting several years. After persisting for six months or a year the symptoms may disappear. There are

remarkable instances in which the symptoms have come on with great intensity, following fright, and have disappeared again in a few days.

Prognosis.—Statistics are misleading as only the severe cases come under hospital treatment. Sattler estimates the mortality at 11 per cent. In Hale White's series it was 84 in 214 cases. In the hands of Kocher and the Mayos the mortality from operation is below 4 per cent. and some 70 per cent. of cases are claimed to have been cured.

Diagnosis.—Few diseases are so easily recognized. The difficulty is with the partially developed forms, *formes frustes*, which are not uncommon. The nervous state, the tremor, and tachycardia may be the only features, or there may be slight swelling of the thyroid with tremor alone. The greatest difficulty arises in the cases of hysterical tremor with rapid heart action. Doubtful cases may be tested by the careful administration of iodine internally, as patients with hyperthyroidism show a marked intolerance, even to small doses. A differential count of the leucocytes shows an increase in the mononuclears.

Treatment.—(*a*) The disease is serious enough to warrant strong measures systematically carried out; much valuable time is lost in trying various remedies. The patient should be in bed, at absolute rest, and see very few persons. To quiet the heart's action the icebag may be continuously applied through the day, and ~~veratrum viride,~~ aconite, or strophanthus given in full doses. Ergot, belladonna, phosphate of soda, small doses of opium, and many other remedies are recommended, and in some instances ~~I have seen benefit from the~~ belladonna and ~~the~~ phosphate of soda. Electricity may be helpful.

(*b*) *Serum Therapy.*—Two methods are employed: feeding with the milk of dethyroidized goats, introduced by Lanz, which is obtainable as a substance called rodagen. Good results have been reported by Mackenzie and others. Beebe, on the other hand, uses the serum of animals into which human thyroid extract has been injected. Excellent results have been obtained, but the method has the danger associated with the use of foreign sera.

(*c*) *Surgical Treatment.*—Removal of part of the thyroid gland offers the best hope of permanent cure. It is remarkable with what rapidity all the symptoms may disappear after partial thyroidectomy. A second operation may be necessary in severe cases. The results obtained by the brothers Mayo and by Kocher give a remarkable percentage of recoveries. The operation under cocaine may be done with safety when the condition of the heart and the extreme tachycardia do not contraindicate it. Tying of the arteries and exothyropexia are also recommended. Excision of the superior cervical ganglia of the sympathetic has one beneficial result, viz., the production of slight ptosis, which obviates the staring character of the exophthalmos.

Marked benefit has followed the use of the X-rays in a few cases.

IV. DISEASES OF THE PARATHYROID GLANDS

The parathyroid bodies occur, as a rule, in two pairs on either side of the lateral lobes of the thyroid gland; small ovoid structures from 6 to 8 mm. in length. Some observers regard them as simply supplementary to the thyroid, without a special function; others believe that they have an important internal secretion supplementing that of the thyroid gland and controlling calcium metabolism.

Tr. for G.D. Ochsner.

equid. avoid excitement or irritation. Lung alcoh. rest w/day
Antipyretic. notably oral chol. – very little meat. (2-3 meals a week max.)
use milk + milk foods. butter n. no meat broths
many vegs. fruits – esp. butter, fresh raw cereals
bulk spoils. If not gas used is little w/milk

The studies of Gley, Halsted, and others leave no question as to the importance of these glands. Following their removal in animals there are twitching, spasms of the voluntary muscles, gradual paralysis with dyspnœa, and death from exhaustion. These sometimes disappear when a saline extract of the parathyroid is injected into a vein, or if the parathyroid glands are fed or transplanted. The association of tetany with the disturbance of the function of the thyroid seems to be definitely established. MacCallum has shown the importance of the function of these glands in controlling calcium metabolism, and it is possible that in impoverishment of the tissues in this ingredient is to be sought the cause of the great excitability of the nervous system and of tetany.

These studies have thrown great light upon various spasmodic disorders of children, and some have gone so far as to embrace such conditions as laryngismus, infantile convulsions, and tetany under the term "spasmophilia" (Heubner).

These glands have also hormonic relations, as yet not thoroughly understood, with the other ductless glands, and have some influence on carbohydrate metabolism.

The definite association of the glands with tetany is sufficient warrant for treating this disease here.

TETANY

Definition.—An affection characterized by bilateral, chronic, or intermittent spasms of the extremities, with gradually increasing irritability of the nerves.

Etiology.—The following groups are made by Frankl-Hochwart:

(*a*) Tetany of Adults.—(1) Epidemic tetany, also known as rheumatic tetany, idiopathic workman's tetany, or shoemaker's cramp. In certain parts of the Continent of Europe the disease has prevailed widely, particularly in the winter season. Von Jacksch, who has described an epidemic form occurring in young men of the working classes, sometimes with slight fever, regards the disease as infectious. This form is acute, lasting only two or three weeks, and rarely proving fatal.

(2) Tetany of gastric and intestinal disorders, as dyspepsia, gastrectasis, diarrhœa, and helminthiasis. The form associated with dilatation of the stomach is rare, not more than 30 cases having been reported.

(3) Tetany of the acute infectious diseases (typhoid, cholera, influenza, measles, scarlatina, etc.). In some typhoid epidemics many cases have occurred.

(4) Tetany following poisoning from chloroform, morphia, ergot, lead, alcohol, and uræmia. Isolated examples of each have been reported.

(5) Tetany may also develop during pregnancy or recur in successive pregnancies. From its occurrence in nursing women, Trousseau called it "nurse's contracture."

(6) Tetany following removal of the thyroid gland is probably due to a removal of the parathyroid bodies at the same time.

(7) Tetany may complicate other nervous disorders, as Basedow's disease, cerebral tumor, cysts of the cerebellum, and syringomyelia.

(*b*) Tetany in Children.—Tetany bears a definite relation to gastro-intestinal disorders, acute infections, and rickets in childhood.

Mild cases of tetany are not uncommon in children, particularly in connection with rickets and gastro-intestinal disorders. The other forms are not common, either in the United States or in England. Campbell Howard reported from my clinic 8 cases of tetany, 4 accompanying dilatation of the stomach, 2 with hyperacidity without dilatation, 1 with chronic diarrhœa, and 1 occurring in connection with repeated pregnancies and lactation.

Morbid Anatomy.—~~It is now well established that~~ the tetany following extirpation of the thyroid gland is due to coincident removal of the parathyroids; and the observations of MacCallum suggest that, occurring spontaneously, it is associated with insufficiency of the parathyroids, which appear to control calcium metabolism. Absence or perversion of the parathyroid secretion leads to impoverishment of the tissues in calcium, and to hyperexcitability of the nerves.

Symptoms.—In cases associated with general debility or in children with rickets the spasm is limited to the hands and feet. The fingers are bent at the metacarpo-phalangeal joint, extended at the terminal joints, pressed close together, and the thumb is contracted in the palm of the hand. The wrist is flexed, the elbows are bent, and the arms are folded over the chest. In the lower limbs the feet are extended and the toes adducted. The muscles of the face and neck are less commonly involved, but in severe cases there may be trismus, and the angles of the mouth are drawn out. The skin of the hands and feet is sometimes tense and œdematous. The spasms are usually paroxysmal and last for a variable time. In children the attack may pass off in a few hours. In some of the more severe chronic cases in adults the stiffness and contracture may continue or even increase for many days, and the attack may last as long as two weeks. In the acute cases the temperature may be elevated and the pulse quickened. In the severe paroxysms there may be involvement of the muscles of the back and of the thorax, inducing dyspnœa and cyanosis. Certain additional features, valuable in diagnosis, are present.

Trousseau's symptom: "So long as the attack is not over, the paroxysms may be reproduced at will. This is effected by simply compressing the affected parts, either in the direction of their principal nerve trunks or over their blood vessels, so as to impede the venous or arterial circulation."

Chvostek's symptom is shown in the remarkable increase in the mechanical excitability of the motor nerves. A slight tap, ~~for example,~~ in the course of the facial nerve will throw the muscles to which it is distributed into active contraction. Erb has shown that the electrical irritability of the motor nerves, especially to the galvanic current, is also greatly increased, and Hofmann has demonstrated the heightened excitability of the sensory nerves, the slightest pressure on which may cause paræsthesia in the region of distribution.

Diagnosis.—The disease is readily recognized. It is a mistake to call instances of carpo-pedal spasm of children true tetany. It is common to find in rickety children or in cases of severe gastro-intestinal catarrh a transient spasm of the fingers or even of the arms. By many authors these are considered cases of mild tetany, and there are all grades in rickety children between the simple carpo-pedal spasm and the condition in which the four extremities

are involved; but it is well, I think, to limit the term *tetany* to the more severe affection.

With true tetanus the disease is scarcely ever confounded, as the commencement of the spasm in the extremities, the attitude of the hands, and the etiological factors are very different. Hysterical contractures are usually unilateral.

Treatment.—In the case of children the condition with which the tetany is associated should be treated. Baths and cold sponging are recommended and often relieve the spasm as promptly as in child-crowing. Bromide of potassium may be tried. In severe cases chloroform inhalations may be given. Massage, electricity, and the spinal icebag have also been used with success. Cases, however, may resist all treatment, and the spasms recur for many years. The thyroid extract should be tried. Gottstein reports relief in a case of long standing, and Bramwell reports one case of operative tetany and one of the idiopathic form successfully treated in this way.

In gastric tetany, especially when due to dilatation of the stomach, the mortality is high, and recovery without operative interference is rare: of 27 cases collected by Riegel, 16 terminated fatally. Cunningham collected 8 cases treated surgically, with a mortality of 37.5 per cent., as compared with 70 per cent. treated by medical means. Regular, systematic lavage with large quantities of saline or mildly antiseptic solutions is sometimes beneficial.

The administration of calcium is frequently effective. It may be given as calcium lactate in doses of 15 grains (1 gm.) every three or four hours. In severe cases much larger amounts may be given.

V. DISEASES OF THE SPLEEN

1. GENERAL REMARKS.

Though a ductless gland, the spleen is not known to have an internal secretion, and its functions are as yet ill understood. It is not an organ essential to life. In the fetus it takes part in the formation of the red blood corpuscles, and as it contains hæmatoblasts, it is possible that in the adult this function may be exercised to some extent, particularly in cases of severe anæmia.

Hæmolysis is generally believed to be its special function, a view—not held by all physiologists—based upon the presence of a large percentage of organic compounds of iron, the deposit in the organ of blood pigments in various diseases, the presence of many macrophages containing red blood corpuscles, and upon the evidence, after removal of the spleen, of compensatory hæmolysis in many newly formed hæmo-lymph glands (Warthin).

Removal of the spleen, an operation practised by the ancients in the belief that it improved the wind of runners, is not, as a rule, followed by serious effects. There may be slight eosinophilia and temporary anæmia, and later there is usually slight leucocytosis, with relative increase of the lymphocytes.

In infections the organ enlarges and micro-organisms are present in large numbers. It has been supposed to play some part in the processes of immunity and phagocytosis goes on actively in the organ. In experimental anæmia caused by various hæmolytic agents the spleen enlarges, and in these conditions Bunting and Norris found evidence of vicarious blood formation. Chronic

enlargement of the spleen may be present with very little disturbance of health. Attacks of anæmia sooner or later occur, and consecutive fibrosis may occur in the liver with jaundice and ascites (Banti's disease).

2. MOVABLE SPLEEN

Movable or wandering spleen is seen most frequently in women the subjects of enteroptosis. It may be present without signs of displacement of other organs. It may be found accidentally in individuals who present no symptoms whatever. In other cases there are dragging, uneasy feelings in the back and side. All grades are met with, from a spleen that can be felt completely below the margin of the ribs to a condition in which the tumor-mass impinges upon the pelvis; indeed, the organ has been found in an inguinal hernia! In the large majority of all cases the spleen is enlarged. Sometimes it appears that the enlargement has caused relaxation of the ligaments; in other instances the relaxation seems congenital, as movable spleens have been found in different members of the same family. Possibly traumatism may account for some of the cases. Apart from the dragging, uneasy sensations and the worry in nervous patients, wandering spleen causes very few serious symptoms. Torsion of the pedicle may produce a very alarming and serious condition, leading to great swelling of the organ, high fever, or even to necrosis. A young woman was admitted to ~~my colleague~~ Kelly's ward with a tumor supposed to be ovarian, but which proved to be a wandering, moderately enlarged spleen. She was transferred to the medical ward, where she had suddenly very great pain in the abdomen, a large swelling in the left flank, and much tenderness. Halsted operated and found an enormously enlarged spleen in a condition of necrosis, adherent to the adjacent parts and to the abdominal wall. He laid it open freely, and large necrotic masses of spleen tissue discharged for some time. She made a good recovery.

The *diagnosis* of a wandering spleen is usually easy unless the organ becomes fixed and is deformed by adhesions and perisplenitis. The shape of the organ and the sharp margin with the notches are the points to be specially noted.

The *treatment* ~~of the condition~~ is important. Occasionally the organ may be kept in position by a properly adapted belt and a pad under the left costal margin. Removal of the displaced organ has been advised and carried out in many cases, and nowadays it is not a very serious operation. It is, however, as a rule unnecessary. In two cases of enlarged spleen under my care, with great mobility, causing much discomfort and uneasiness, Halsted completely relieved the condition by replacing the spleen, packing it in position with gauze, and allowing firm adhesions to take place. Both these patients were seen more than eighteen months after the operation and the organ had remained in position.

3. RUPTURE OF THE SPLEEN

This is of interest in connection with the spontaneous rupture in cases of acute enlargement during typhoid fever or malaria, which is very rare. Rupture of a malarial spleen may follow a blow, or a fall, or an exploratory puncture. In India and in Mauritius rupture of the spleen is stated to be very

common. Fatal hæmorrhage may follow puncture of a swollen spleen with a hypodermic needle. Occasionally the rupture results from the breaking of an infarct or of an abscess. The symptoms are those of hæmorrhage into the peritoneum, and the condition demands immediate laparotomy.

4. INFARCT, ABSCESS AND CYSTS OF THE SPLEEN

Emboli in the splenic arteries causing *infarcts* may be either infective or simple. They are seen most frequently in ulcerative endocarditis and in septic conditions. Infarcts may also follow the formation of thrombi in the branches of the splenic artery in cases of fever. They are not very infrequent in typhoid. In a few instances the infarcts have followed thrombosis in the splenic veins. They are chiefly of pathological interest. The infarct of the spleen may be suspected in cases of septicæmia or pyæmia when there are pain in the splenic region, tenderness on pressure, and slight swelling of the organ; ~~on several occasions~~ I have heard a well-marked peritoneal friction rub. Occasionally in the infective infarcts large *abscesses* are formed, and in rare instances the whole organ may be converted into a sac of pus.

Tumors of the spleen, hydatid and other *cysts* of the organ, and *gummata* are rare conditions of anatomical interest. In Hodgkin's disease the organ may be enlarged and smooth, or irregular from the presence of nodular tumors.

Cysts are rare; I have seen but ~~two~~, one an echinococcus, and the other a double cyst of the hilus. The latter apparently are ~~very~~ common, and probably arise from a hæmatoma subcapsular or in the hilus. They have been successfully removed. Very small cysts are not infrequent in connection with polycystic disease of the liver and the kidneys. A dermoid cyst has been described. The diagnosis of cysts is not often made; the mass is usually irregular in the region of the spleen, but the splenic outlines are marked. In the case ~~I saw~~ with two cysts at the hilus, the tumor was very movable, ~~very~~ and irregular, ~~and I~~ urged operation on the grounds of mechanical discomfort, and increase in size. ~~In a recent paper~~ Musser stated that there have been 21 operations, all successful, ~~in cysts of this sort~~.

5. PRIMARY SPLENOMEGALY WITH ANÆMIA

(Splenic Anæmia, Banti's Disease)

Definition.—A primary disease of the spleen of unknown origin, characterized by progressive enlargement, attacks of anæmia, a tendency to hæmorrhage, and in some cases a secondary cirrhosis of the liver, with jaundice and ascites. That the spleen itself is the seat of the disease is shown by the fact that complete recovery follows its removal.

History.—The name "splenic anæmia" was applied to a group of cases by Griesinger in 1866. H. C. Wood, in 1871, described cases as the splenic form of pseudo-leukæmia. The real study of the disease was initiated by Banti in 1883. In France the condition was called "primitive splenomegaly," and many different types ~~of the disease~~ have been described. Here we shall deal only with the form referred to in the definition as splenic anæmia and Banti's disease.

Etiology.—In the majority of cases the enlargement of the spleen comes

on without any recognizable cause. In a few of my cases malaria has been present, but in the greater number the first thing noticed has been the mechanical inconvenience of the big spleen. Males are more frequently attacked than females. It is a disease of young and middle life, the majority of cases occurring before the fortieth year. It is also met with in young children. Many of the cases of infantile splenic anæmia of von Jaksch and of the Italian writers belong to this disease.

Morbid Anatomy.—The spleen is greatly enlarged, coming perhaps next to the size to the leukæmic organ. It is very firm, the capsule is thickened, the texture of the gland very tough and firm, and the whole in a state of advanced fibrosis. Banti has described a proliferation of the endothelial cells of the venous sinuses of the pulp, and he believes there are very characteristic histological changes.

The blood vessels in the neighborhood of the spleen may be very large, particularly the vasa brevia, and the splenic vein itself and the portal vein may be enormously dilated, and show atheroma and calcification. The lymphatic glands are not involved. Hyperplasia of the bone marrow has been found, but no other changes of special importance.

The cases of the Gaucher type, primitive endothelioma of the spleen, do not belong in this group.

Symptoms.—The disease is extraordinarily chronic; seven of my cases had a longer duration than ten years. Usually the first feature to attract attention is:

Splenomegaly.—The enlargement is uniform, smooth, painless, usually reaches to the navel, very often to the anterior superior spine, and the organ may occupy the whole of the left half of the abdomen. It may exist for years without any symptoms other than the inconvenience caused by the distention of the abdomen. Following an infarct pain may be present.

Anæmia.—Sooner or later the patients become anæmic. The attack may develop with rapidity, and in children a severe and even fatal form may follow in a few weeks. More commonly the pallor is gradual and the patient may come under observation for the first time with swelling of the feet, shortness of breath, and all the signs of advanced anæmia. The blood picture is, as a rule, that of a secondary anæmia with a very low color index and a marked leucopenia. The red blood corpuscles may fall as low as two million and in an average of a series of uncomplicated cases the leucocyte count was under 3,500 per c. mm. There are no special changes in the differential count. Following a severe hæmorrhage there may be a rise in the leucocytes. Some patients have permanent slight anæmia of the secondary type; others remain very well except for recurring attacks of anæmia, of great severity, which may be independent of hæmorrhage.

The question whether the anæmia splenica infantum of von Jaksch and the Italian writers is the same malady has been much discussed. There are cases in which enlargement of the spleen without obvious cause has been followed by a rapidly progressing anæmia with marked leucopenia. A very suggestive thing is that in a case in a child, with a blood count below one million reds, removal of the spleen was followed by complete recovery.

Hæmorrhages.—Bleeding, usually hæmatemesis, may be a special feature of the disease and may occur for many years. One of my patients had recurring

chron. adh. complete granular or vacuolated - hyperdermatosis

Jackson. J. A. M. A. Bull. 1916, XXVII, 1

Th. other infantile.

19 cases. 15 females. 11 had hemorrhage (nose, gums, skin)

10 had abd. pain.

Spleen. enlarged -

Liver. enlarged in 14. take place gradually than spleen.

glands - sm. enlarged

pig. - in most efficient

Conjunctival thickening. in some

blood - sec. an. temporarily low.

Splenectomy - in 9. 6 in no great diff.

Syph. + tuberc. - not examined -

Path. ? spleen, liver

gl. thymus

adrenals

bone marrow.

Blood films in fatal cases

attacks for twelve years, and one at the London Hospital, noted by R. Hutchison, for fifteen years. In such cases the diagnosis of ulcer of the stomach may be made. The bleeding may be of great severity. On several occasions one of my patients was brought into the hospital completely exsanguine; in two the hæmorrhage proved directly fatal; in a third the hæmorrhage proved fatal ten days after a successful removal of the spleen. The bleeding comes, as a rule, from œsophageal varices. Melæna may be present. Hæmaturia occurred in one of ~~my~~ cases; purpura is not uncommon.

Ascites.—Usually a terminal event, it may be due to the enlarged spleen itself or to secondary cirrhosis of the liver. When due to the liver, it is associated with slight jaundice.

Jaundice.—Icterus has been a rare symptom in ~~my~~ cases. Enlargement of the spleen may persist for many years without any consecutive change in the liver. One patient ~~has~~ had splenomegaly ~~with~~ repeated hæmorrhages and ~~had now (1912)~~ had more than twelve years of good health after splenectomy. Slight jaundice may persist for years, sometimes with enlargement of the liver, in others with distinct reduction in its volume, and in either case with a progressive cirrhosis—the features to which Banti called special attention.

Course of the Disease.—It is extraordinarily chronic. A patient may for ten or twelve years have a large spleen causing no inconvenience, then an attack of anæmia may occur, from which recovery gradually takes place; or the first symptom may be ascites or a severe hæmorrhage from the stomach. As a rule, the anæmia becomes more or less chronic, with marked exacerbations, and in the later stages jaundice with ascites develops.

Diagnosis.—Here may be mentioned a series of forms of splenomegaly which differ essentially from the splenic anæmia, and in which, so far as we know, the condition of the spleen is not primary.

Splenomegaly with Acholuric Jaundice.—This type, first described by Minkowski and sometimes called after his name, is usually a familial form, often hereditary. It is consistent with good health throughout life, and there may be no symptoms. Characteristic features are: (*a*) its familial form; (*b*) chronic enlargement of the spleen; (*c*) good health; (*d*) chronic slight jaundice; (*e*) presence of urobilin in the urine, but absence of bile pigment. In a few instances gall stone colic has been present, due to the presence of small calculi. Chauffard showed that the red blood corpuscles in these cases have an increased fragility, the cause of which is unknown, but this is an essential feature in the hæmolytic jaundice. In the familial form good health is the rule, but in the acquired form the patient often becomes anæmic and is very ill. Cures have been reported after splenectomy.

Splenomegaly of the Gaucher Type (*Primary Endothelioma of the Spleen*).—In a dozen or more cases the enlarged spleen has retained its form and presents on section a grayish red appearance, with whitish spots or streaks; but histologically shows large cells from 20 to 40 μ in size, filling the alveolar spaces. The nature of this affection, first described by Gaucher in 1882, has been much discussed. The general opinion now is that of Stengel, that it is a type of new growth, an endothelioma; in any case, it differs entirely from the splenic anæmia.

Splenomegaly Associated with Primary Pylethrombosis.—Cases have been reported of enlarged spleen in connection with phlebitis of the

splenic and portal veins, and clinically such cases closely resemble Banti's disease. The spleen is very large and there are jaundice and ascites with moderate anæmia. The recognition of the pylethrombosis is only made post mortem.

Hepatic Splenomegaly.—Three varieties of cirrhosis of the liver may lead to great enlargement of the spleen with anæmia and a symptom-complex resembling that of splenic anæmia.

(*a*) *Alcoholic Cirrhosis.*—With recurring hæmorrhages, a consecutive anæmia, ascites, and an unusually large spleen, the condition may simulate closely the last stage of splenic anæmia. The history, particularly the late appearance of the hepatic changes, may be the most important point. In the cases in which I have been in doubt the difficulty has arisen from an imperfect history and from the presence of recurring hæmorrhages.

(*b*) *Syphilitic Cirrhosis.*—Great enlargement of the spleen may follow gummous hepatitis, either congenital or acquired. Toward the close the picture is very similar to Banti's disease—slight jaundice, ascites, big spleen, recurring hæmorrhages, and marked anæmia. Signs of other syphilitic lesions and the irregular nodular liver may suggest the diagnosis.

(*c*) In a few cases of hypertrophic cirrhosis, as in Hanot's form and in hæmochromatosis, the spleen may be greatly enlarged, and in the late stages, when ascites and hæmorrhages occur, the clinical picture may be like that of splenic anæmia.

Splenomegaly in Pernicious Anæmia.—Sometimes the spleen is greatly enlarged in this disease, reaching to the navel, but, as a rule, the lower blood count, the high color index, the large number of nucleated red blood corpuscles, and the clinical course enable one to make the diagnosis.

Tropical Splenomegaly.—Kala-azar has been considered elsewhere and can be distinguished by the presence of the Leishman-Donovan bodies in the splenic blood. There are big spleens with anæmia in the Tropics which are not Kala-azar, and the experience of some of the physicians in Cairo indicates that some of these, at any rate, are of the ordinary splenic anæmia type, in which removal of the organ cures the disease.

Treatment.—In the first stage with simple splenomegaly nothing is indicated; the patients, as a rule, look very well. For the anæmia the usual measures may be adopted, and the patient gets gradually better. The ultimate outlook is bad, and there is only one radical cure—removal of the spleen. This has now been done in a sufficiently large number of cases to determine its value. Of 6 of my cases 3 have recovered; one is alive more than 12 years after the operation, another between 6 and 7. One died on the table from hæmorrhage, a second from shock, and a third died ten days after from a rupture of the œsophageal varix. Armstrong of Montreal has collected 32 cases operated upon with 9 deaths. The fact that removal of the spleen is followed by complete recovery, even after the appearance of the jaundice and of chronic anæmia, is the best proof that the source of the trouble is in this organ itself, and is one of the best warrants for the recognition of the disease as a separate clinical entity.

Pituitary + carb.

over activity of post. lobe (pars nervosa + infundibular) favors glycogenolysis - gives lowered sugar tol. - hyperglycemia

less activity or hypoplasia - carb. tol. becomes higher - post. lobe extract by inhalation - assistant. injection lowers sugar tolerance + may cause glycosuria.

acromegaly - early stages - sugar tolerance low - but later an acquired ovartolerance.

high assimilation limit may be expected when gland is compressed or obstructed by superimposed tumor which acts like a posterior lobe hypoplasia

Early acromegaly in early stage gland when therapy surgical is indicated.

If increased carb. tol. apt to have adiposity.

Blumenthal argues that sugar ad. begiven subcutaneously to increase

J.A.M.A. Lundgaard, Sa[illegible] + Wilder - Dec. 11. 1915. have [illegible] applied to[illegible] fluid + health - resting - that sugar caused it .8 - .99 - per kilo body weight: patients Therefore pit. cases to show normal tolerance

VI. DISEASES OF THE PITUITARY BODY

The hypophysis cerebri consists of two lobes, (*a*) an anterior lobe, originating from the roof of the pharynx and composed of large granular epithelial cells arranged in columns surrounded by large venous spaces into which their secretion discharges; and (*b*) a smaller posterior lobe which arises from the floor of the third ventricle and is composed (1) of a central neuroglial portion (pars nervosa) and (2) an investment of epithelial cells (pars intermedia). The secretion of the posterior lobe is supposed by some to find its way into the cerebro-spinal fluid.

Complete experimental removal of the gland is fatal (Paulesco). Partial removal leads, in young animals, to a stunting of growth, to adiposity and failure of sexual development, in adult animals to adiposity and genital dystrophy (Cushing).

Modern knowledge of the functions of the gland began with the studies of Marie on the relation of the pituitary gland to acromegaly and gigantism. Then Schäfer and Oliver discovered that injection of an extract of the gland caused a rise in the blood pressure. Since these two cardinal observations an enormous amount of work has been done, and we are beginning to appreciate the remarkable influence of this small structure upon the processes of development and metabolism. Briefly, the anterior lobe influences growth and development, and is necessary to life; the posterior lobe influences the metabolism of the carbohydrates and fats.

Disturbances in the function of the pituitary gland are not clearly grouped, as in the thyroid, into the effects of deficiency and excess, though one can usually differentiate states of hyper- and hypopituitarism. The hypophysis appears to be closely related to other glands of internal secretion and, as is well known, involvement of any member of the series causes a physiological readjustment in the activity of the others. Owing to the situation of the gland it is very liable to feel the effect of pressure in neighboring or even in distant lesions, so that disturbance of function may be due not only to a primary involvement, but to secondary compression. As a result of his experimental work and studies of clinical cases Cushing prefers to group the conditions associated with disturbance of the function of the gland under the term "dyspituitarism" and recognizes a number of groups:

(*a*) Cases of tumor growth showing signs of distortion of neighboring structures, and also the constitutional effects of altered glandular activity. The X-rays show changes in the configuration of the pituitary fossa; there are pressure signs on the adjacent cranial nerves, bi-temporal hemianopia, optic atrophy, and occulomotor palsies. Uncinate fits are not unusual. Epistaxis is common and cerebro-spinal rhinorrhœa may occur in rare cases. The constitutional effects vary from primary over-activity to glandular underactivity.

(*b*) Cases in which the neighborhood manifestations are pronounced but the constitutional features are slight. The characteristic regional signs of tumor are marked, but there may be slight or very transient evidence of disturbed glandular activity, perhaps only disturbed carbohydrate metabolism with adiposity.

(*c*) Cases in which the neighborhood manifestations are absent or slight, though the glandular symptoms are unmistakable. The gland is not so large as to cause regional symptoms. There are skeletal changes on the side of overgrowth or undergrowth. Disturbance of carbohydrate metabolism is a matter of modified posterior lobe activity, whether occurring as a lowering of the assimilation limit, which is so often associated with the early stages of acromegaly, or a great increase in tolerance, such as characterizes all grades of hypopituitarism. In posterior lobe insufficiency there are a tendency to the deposition of fat, a subnormal temperature, drowsiness, slow pulse, dry skin, loss of hair, and an extraordinary high tolerance for sugars. Most of the cases of acromegaly fall in this group and show at first evidences of hyperpituitarism, and later of insufficiency. In the adult, adiposity, high sugar tolerance, subnormal temperature, psychic manifestations, and sexual infantilism of the reversive type indicate hypopituitarism and may exist without the regional symptoms of tumor (Cushing).

(*d*) Hypophysial symptoms may be shown by patients with internal hydrocephalus from any cause, probably by interference with the passage of the posterior lobe secretion into the cerebro-spinal fluid, and this obstructive dyspituitarism may result from any lesion, inflammatory or neoplastic, in the neighborhood of the third ventricle.

These are the most important of the groups to which Cushing refers, but there are also cases with manifestations indicating involvement of other internal secretions together with that of the hypophysis, and a large group in which transient hypophysial symptoms occur, as, in pregnancy, cranial injuries and infectious diseases.

It is quite clear that disturbances in the function of the pituitary gland may lead to remarkable changes in growth; *hyperpituitarism* may lead to gigantism, when the process antedates ossification of the epiphyses—the Launois type; to acromegaly when it is of later date; *hypopituitarism* to adiposity, with skeletal and sexual infantilism when the process originates in childhood—the Frölich type; to adiposity and sexual infantilism of the reversive type when originating in the adult.

Much has been done to clear the subject, but much remains, particularly to clear up the relations of the various types of infantilism which have been described—the Lorain, the Brissaud, the pancreatic, the intestinal—to the different internal secretions. One condition is important enough to merit separate consideration, the one differentiated clearly by Marie and known as acromegaly. (The student is referred to Hastings Gilford's "Disorders of Post-natal Growth," to Vincent's "Innere Secretion," *Ergeb. d. Phys.*, IX and X, and to Cushing's work, "The Pituitary Gland and Its Disorders," J. B. Lippincott Co., 1912.)

ACROMEGALY

Definition.—A dystrophy characterized by increase in size of the face and extremities associated with perverted function of the pituitary gland.

The essence of the disease is a hyperpituitarism which, if it antedates ossification of the epiphyses, leads to gigantism, and in the adult leads to overgrowth of the skeleton and other changes which we know as acromegaly.

Etiology.—It is a rare disease, and rather more frequent in women. It

affects particularly persons of large size. Twenty per cent. of acromegalics are above six feet in height when the symptoms begin, and fully 40 per cent. of giants are acromegalics (Sternberg). Trauma, the infections, and emotional shock have preceded the onset of the disease.

Pathology.—Practically all of the cases show changes in the pituitary gland, hyperplasia, adenoma, fibroma, or sarcoma, causing distention of the sella turcica and, in the late stages, pressure on surrounding structures; the symptoms of the disease are in part due to disturbance of the function of the gland, and in part to the pressure on the adjacent parts.

The bones show the most striking changes; there is a general enlargement of the extremities, but the skeleton on the whole is more or less affected. The enlargement, due to a periosteal growth, is most evident in the hands and feet. The bones of the face are always involved. The orbital arches, frontal prominences, zygoma, malar, and nasal bones are all increased in size, the lower jaw is elongated, thickened, and the teeth separated. The X-ray picture shows very characteristic changes in the sella turcica. The skin and subcutaneous tissues are thickened, so that the enlargement of the extremities is not altogether bony, and the hypertrophy is seen in the soft parts of the face as well.

The brain has been found large, but the most important changes are those due to pressure at the base. The internal organs have been found enlarged, and in Osborne's case the heart weighed 2 lbs. 9 oz.

Symptoms.—As already mentioned, when the pituitary gland is involved in tumor growth, which is the common condition in acromegaly, the symptoms may be grouped into those due to the mechanical effects and those associated with perversion of the secretion of the gland.

(*a*) Regional Symptoms.—Headache is common, usually frontal. Somnolence has been noted in many cases, and in one of my patients was the first symptom. Ocular features occur in a large proportion of the cases, bitemporal hemianopia, optic atrophy, and, in the late stages, pressure on the third nerve and the abducens. One eye only may be affected. Exophthalmos may occur. Deafness is not infrequent. Irritability of temper, marked change in the disposition, great depression, and progressive dementia have been noted. Epistaxis and rhinorrhœa may be present.

(*b*) Symptoms due to the perversion of the internal secretion itself form the striking features of the disease. The patient's friends first notice a gradual increase in the features, which become heavy and thick; or the patient himself may notice that he takes a larger size of hat, or with the progressive enlargement of the hands a larger size of gloves. The enlargement of the extremities does not interfere with their free use.

The hypertrophy is general, involving all the tissues, and gives a curious spadelike character to the hands. The lines on the palms are much deepened. The wrists may be enlarged, but the arms are rarely affected. The feet are involved like the hands and are uniformly enlarged. The big toe, however, may be much larger in proportion. The nails are usually broad and large, but there is no curving, and the terminal phalanges are not bulbous. The head increases in volume, but not as much in proportion as the face, which becomes much elongated and enlarged in consequence of the increase in the size of the superior and inferior maxillary bones. The latter in particular increases

greatly in size, and often projects below the upper jaw. The alveolar processes are widened and the teeth are often separated. The soft parts also increase in size, and the nostrils are large and broad. The eyelids are sometimes greatly thickened, and the ears enormously hypertrophied. The tongue in some instances becomes greatly enlarged. Late in the disease the spine may be affected and the back bowed—kyphosis. The bones of the thorax may slowly and progressively enlarge. With this gradual increase in size the skin of the hands and face may appear normal. Sometimes it is slightly altered in color, coarse, or flabby, but it has not the dry, harsh appearance of the skin in myxœdema. The muscles are sometimes wasted.

Also associated with disturbance of the function of the gland is the diabetes noticed in many cases, which is common in the early stages; in the advanced stages there is an extraordinary high tolerance for sugar. Symptoms on the part of other ductless glands are common. Goitre is of frequent occurrence. Amenorrhœa is the earliest symptom in women. Impotence is common in advanced cases in men.

The various symptoms ~~of the disease~~ are now readily explained with the knowledge we possess of the functions of the gland, which have already been discussed.

Treatment.—The use of extracts of the gland has been extensively tried. Possibly now with our better knowledge of the functions of the different parts we shall arrive at a more intelligent organo-therapy. Unquestionably glandular therapy should only be used when the condition has passed into its ultimate stage of glandular insufficiency—a stage which is indicated usually by an increased sugar tolerance.

Surgical treatment has been carried out in a number of cases following Schloffer's suggestion. Doubtless the chief surgical indication is to give relief to the local pressure symptoms when there is marked glandular enlargement. The tumor or glandular struma may be reached by a transphenoidal or a subtemporal route. Partial removal of the growth or the evacuation of a cyst under favorable circumstances may save the optic nerves from complete pressure atrophy.

VII. INFANTILISM

Definition.—The failure of the appearance of the primary and secondary sexual characteristics, together with the retention of mental and bodily conditions of childhood.

Etiology.—It is not possible at present to make a satisfactory classification either of the causes or of the cases of infantilism—in some no cause is evident, in others the failure in development has followed obvious disease, and there are cases directly dependent upon loss of some internal secretion.

I. Cachectic infantilism is by no means uncommon, as any serious chronic malady may delay sexual development. For example, the children affected with hookworm disease may reach the age of 20 or older before the change from the infantile to the adult state. Syphilis is a very common cause. In regions in which malaria is very prevalent delayed sexual development is not uncommon in children, and we see it not infrequently in cases of con-

genital heart disease. There is also a toxic infantilism due to the slow and prolonged action of alcohol and tobacco.

II. Idiopathic Infantilism (*So-called Lorain Type*).—"In this variety the figure is so small that, at first sight, it looks like that of a child. When the patient is stripped, however, his outlines are seen to be those of an adult, and not those of childhood. The head is proportionately small, and the trunk well formed; for the shoulders are broad compared to the hips, and the bony prominences and the muscles stand out distinctly. We have before us a miniature man (or woman, as the case may be), and not one who has retained the characteristics of childhood beyond the proper time. There is, indeed, no growth of facial, pubic or axillary hair, yet the genital organs, though small, are well shaped and quite large enough for the size of the body. The intelligence in both sexes is generally normal" (John Thomson).

The cause of this form is yet unknown, but it is probably associated with perversion of the pituitary secretions. It has also been called an "angioplastic infantilism," in the belief that it was due to a defect of development of the vascular system.

III. The Hormonic Type.—Here we are on safer ground, as we know definitely of several varieties directly dependent upon changes in the ductless glands. The most important of these are:

(*a*) Thyroidal or Cretinoid Infantilism.—This form has already been described.

(*b*) The Frölich type, dystrophia adiposo-genitalis, associated with a tumor of the pituitary region, is characterized by great obesity and genital hypoplasia. The symptoms are due to a secretory deficit, for they are capable of experimental reproduction by partial glandular extirpation in animals (Cushing). There are adult and infantile types, just as there are in myxœdema; in the former the individual becomes fat and the sexual organs revert to the preadolescent state. The *Brissaud type* is in all probability due to hypopituitarism. A round, chubby face, under-developed skeleton, prominent abdomen, large layer of fat over the whole body, rudimentary sexual organs, no growth of hair except on the head, and absence of the second dentition, are some of the prominent features of this form, which Brissaud attributed to hypothyroidism, but which appears more likely to be due to dyspituitarism.

(*c*) Pancreatico-intestinal Type.—Bramwell, Herter, Freedman, and others have reported cases of infantilism associated with intestinal changes. Bramwell thought the pancreas was at fault, and his cases improved remarkably under treatment with pancreatic extract. In Herter's case there were looseness of the bowels, often fatty stools, and a change in the flora of the intestine with a rise in the ethereal sulphates in the urine.

IV. Progeria.—Under this term Hastings Gilford has described a condition in children of incomplete development (infantilism) with premature decay. The facial appearance, the attitude, the loss of hair, wasting of the skin, are those of old age, and post mortem the most extensive fibroid changes in the organs, particularly in the arteries and kidneys. The condition is probably associated with unknown changes in the internal secretions.

SECTION XI

DISEASES OF THE NERVOUS SYSTEM

A. GENERAL INTRODUCTION

The Neurone.—Its Structure.—The nervous system is a combination of an immense number of units called neurones and all having an essentially similar structure. Each is composed of a receptive cell body and of conducting elements—namely, protoplasmic processes or dendrites, and the axis-cylinder process or axone. The dendrites conduct impulses toward the cell body (cellulipetal conduction) and the axones conduct them away from the cell (cellulifugal conduction). Depending upon whether the axones conduct impulses in a direction away from or toward the cerebrum they are called efferent or afferent. The axis-cylinder process gives off at varying intervals lateral branches called collaterals, running at right angles to the process, and these, and finally the axis-cylinder process itself, split up at their terminations into many fine fibres, forming the end brushes. These, known as arborizations, surround the body of one or more of the many other cells, or interlace with their protoplasmic processes. Thus, the terminals of the axone of one neurone are related to the dendrites and cell bodies of other neurones by contact or by concrescence.

Function of the Neurone.—As already stated, the function of the neurone is to conduct nervous impulses. Reduced to its simplest form, the mode of action may be represented by two cells, one of which, reacting to the environment, conducts impulses inward, whereas the other, awakened by this afferent impulse, conducts an impulse outward. This reflex response Marshall Hall showed to be the fundamental principle of action of the nervous system. The environment acts on the afferent neurones through special sense organs, so that a variety of afferent impulses, olfactory, visual, auditory, gustatory, tactile, painful, thermic, muscular, visceral, and vascular, may be originated. The efferent neurones convey impulses outward to non-nervous tissues, to the skeletal, visceral, and vascular muscles and to the secretory glands, whose activities may thus be augmented or inhibited. The more important reflex centres lie in the bulbo-spinal axis. The situation of the vascular and respiratory centres in the bulb makes it the vital centre of the body. In the spinal cord the location of many reflex centres, particularly those for the muscle tendons and for some of the viscera, is represented in the table on page 898. The visceral mechanism is almost wholly regulated by the bulbo-spinal axis, and its reactions are usually unperceived. Only in conditions of disease do the visceral reflexes "rise into consciousness," and it is at such times that the referred pains and areas of tenderness are produced in the skin-fields of

the spinal segments corresponding to the centre for registration of the visceral reflex.

Degeneration and Regeneration of the Neurone.—The nutrition of the neurone depends in large part upon the condition of the cell body, and this in turn upon the activity of the nucleus. If the cell is injured in any manner the processes degenerate, or the processes separated from the cell degenerate. Though the nerve cells cease to multiply soon after birth, they nevertheless retain remarkable powers of growth and repair. Injury to the cell body may not be recovered from, but if the axone be severed and degeneration take place in consequence, it may under favorable circumstances be replaced by sprouts from the central stump, and its function be regained. Bethe and others believe that the peripheral section, independently of the cell body, has the power of regeneration. It is probable, however, that both factors play a part in the regeneration—namely, the down growth of the axone from the central end of the divided nerve as well as the changes in the periphery, which are most marked in the cells of the sheath of Schwann.

Cell Systems.—The cell bodies of the neurones are collected more or less closely together in the gray matter of the brain and spinal cord and in the ganglia of the peripheral nerves. Their processes, especially the axis-cylinder processes, run for the most part in the white tracts of the brain and spinal cord and in the peripheral nerves. In this way the different parts of the central nervous system are brought into relation with each other and with the rest of the body. Furthermore, the axis-cylinder processes arising from cells subserving similar functions are collected together into bundles or tracts, and though in many cases the course of these tracts and the functions which they possess are extremely complicated and as yet have not been completely unravelled, nevertheless some of them are simple and fairly well understood. By the study of the degenerations that have resulted from injury or from the toxins of certain diseases which possess an affinity for one or another of these individual tracts or systems, it has been possible to trace the course of certain of them through the nervous system. Fortunately for the clinician, the best understood and the simplest system in its arrangement is that which conveys motor impulses from the cortex to the periphery—the so-called pyramidal tract.

The Motor System.—Motor impulses starting in the left side of the brain cause contractions of muscles on the right side of the body, and those from the right side of the brain in muscles of the left side of the body. Leaving out of consideration some few exceptions, it may be stated as a general rule that the motor path is crossed, and that the crossing takes place in the upper segment (Figs. 10 and 11). Every muscular movement, even the simplest, requires the activity of many neurones. In the production of each movement special neurones are brought into play in a definite combination, and acting in this combination specific movement is the result. In other words, all the movements of the body are represented in the central nervous system by combinations of neurones—that is, they are localized. Muscular movements are localized in every part of the motor path, so that in cases of disease of the nervous system a study of the motor defect often enables one to fix upon the site of the process, and it would be hard to over-estimate the importance of a thorough knowledge of such localization. A voluntary motor impulse starting

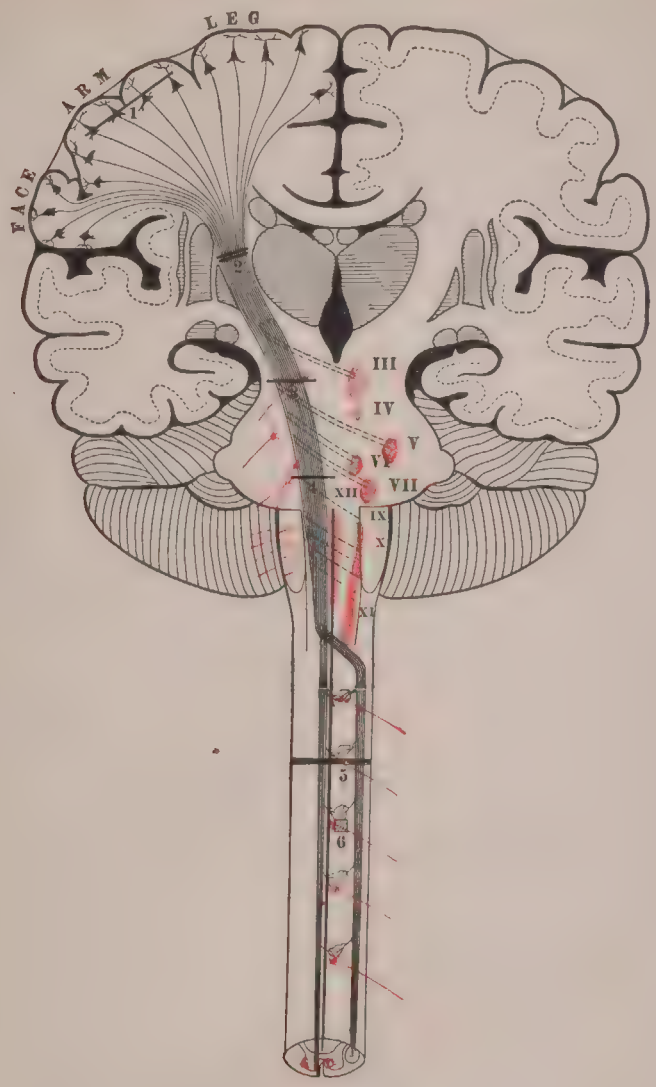


FIG. 10.—DIAGRAM OF MOTOR PATH FROM LEFT BRAIN. The upper segment is black, the lower red. The nuclei of the motor cerebral nerves are shown in red on the right side; on the left side the cerebral nerves of that side are indicated. A lesion at 1 would cause upper segment paralysis in the arm of the opposite side—cerebral monoplegia; at 2, upper segment paralysis of the whole opposite side of the body—hemiplegia; at 3, upper segment paralysis of the opposite face, arm, and leg, and lower segment paralysis of the eye muscles on the same side—crossed paralysis; at 4, upper segment paralysis of opposite arm and leg, and lower segment paralysis of the face and the external rectus on the same side—crossed paralysis; at 5, upper segment paralysis of all muscles below lesion, and lower segment paralysis of muscles represented at level of lesion—spinal paraplegia; at 6, lower segment paralysis of muscles localized at seat of lesion—anterior poliomyelitis. (Van Gehuchten, modified.)

from the brain cortex must pass through at least two neurones before it can reach the muscles, and we therefore speak of the motor tract as being composed of two segments—an upper and a lower.

The Lower Motor Segment.—The neurones of the lower segment have the cell bodies and their protoplasmic processes in the different levels of the ventral horns of the spinal cord and in the motor nuclei of the cerebral nerves. The axis-cylinder processes of the lower motor neurones leave the spinal cord in the ventral roots and run in the peripheral nerves, to be distributed to all the muscles of the body, where they end in arborizations in the motor end plates. These neurones are direct—that is, their cell bodies, their processes, and the muscles in which they end are all on the same side of the body.

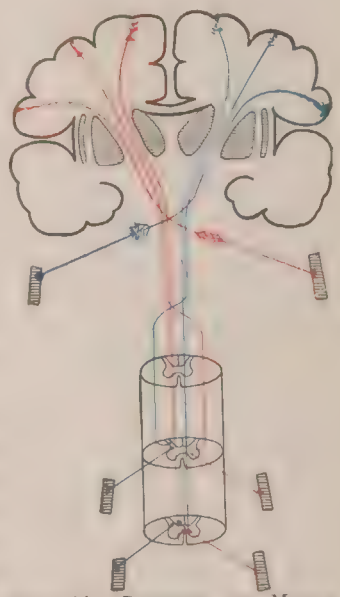


Fig. 11.—Diagram of Motor Path from Each Hemisphere, Showing the Crossing of the Path, Which Takes Place in the Upper Segment Both for the Cranial and Spinal Nerves. (Van Gehuchten, colored.)

The ventral roots of the spinal cord are collected, from above down, into small groups, which, after joining with the dorsal roots of the same level of the cord, leave the spinal canal between the vertebræ as the spinal nerves. That part of the cord from which the roots forming a single spinal nerve arise is called a segment, and corresponds to the nerve which arises from it and not to the vertebra to which it may be opposite. With the exception of the cervical region, in which all the nerve roots but the eighth emerge from above the vertebræ, the roots of each segment for the remainder of the cord leave the spinal canal below the vertebra of corresponding number, and consequently, owing to the fact that during growth the bony canal lengthens much more than the cord itself, the more tailward one goes the greater is the discrepancy in position between each spinal segment and its particular vertebra. This must be borne in mind when determining upon the site of a lesion known to occupy a given segment, for it may lie far above the vertebra of like number and name. A chart has been prepared from numerous measurements by Reid showing the level of the various segments of the cord in relation to the spines of the vertebræ. The axis-cylinder processes which go to make up any one peripheral nerve do not necessarily arise from the same segment of the spinal cord; in fact, most peripheral nerves contain processes from several often quite widely separated segments. Most of the long striped muscles, furthermore, having originated in the embryo from more than one myatome, are innervated from more than one segment.

Our knowledge of the localization of the muscular movements in the gray matter of the lower motor segment is far from complete, but enough is known to aid materially in determining the site of a spinal lesion. The following table, in which is included for each of the spinal segments the centres of recur-

sentation for the more important skeletal muscles, the main reflex centres, and the main location of the segmental skin-field, has been prepared from the studies of Starr, Edinger, Wichmann, Sherrington, Bolk, and others:

LOCALIZATION OF THE FUNCTIONS IN THE SEGMENTS OF THE SPINAL CORD

SEGMENT.	STRIPED MUSCLES.	REFLEX.	SKIN-FIELDS (CF. FIGS. 16 AND 17).
I, II and III C.	Splenius capitis. Hyoid muscles. Sterno-mastoid. Trapezius. Diaphragm (C III–V). Levator scapulæ (C III–V).	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs (diaphragmatic).	Back of head to vertex. Neck (upper part).
IV C.	Trapezius. Diaphragm. Levator scapulæ. Scaleni (C IV–T I). Teres minor. Supraspinatus. Rhomboid.	Dilatation of the pupil produced by irritation of neck. Reflex through the sympathetic (C IV–T I).	Neck (lower part to second rib). Upper shoulder.
V C.	Diaphragm. Teres minor. Supra and infra spinatus (C V–VI). Rhomboid. Subscapularis. Deltoid. Biceps. Brachialis anticus. Supinator longus (C V–VII). Supinator brevis (C V–VII). Pectoralis (clavicular part). Serratus magnus.	Scapular (C V–T I). Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus and biceps. Tapping their tendons produces flexion of forearm.	Outer side of shoulder and upper arm over deltoid region.
VI C.	Teres minor and major Infraspinatus. Deltoid. Biceps. Brachialis anticus. Supinator longus. Supinator brevis. Pectoralis (clavicular part). Serratus magnus (C V–VIII). Coraco-brachialis. Pronator teres. Triceps (outer and long heads). Extensors of wrist (C VI–VIII).	Triceps. Tapping elbow tendon produces extension of forearm. Posterior wrist. Tapping tendons causes extension of hand (C VI–VII).	Outer side of forearm, front and back. Outer half of hand (?).
VII C.	Teres major. Subscapularis. Deltoid (posterior part). Pectoralis major (costal part). Pectoralis minor. Serratus magnus. Pronators of wrist. Triceps. Extensors of wrist and fingers. Flexors of wrist. Latissimus dorsi (C VI–VIII).	Scapulo-humeral. Tapping the inner lower edge of scapula causes adduction of the arm. Anterior wrist. Tapping anterior tendons causes flexion of wrist (C VII–VIII).	Inner side and back of arm and forearm. Radial half of the hand.

LOCALIZATION OF THE FUNCTIONS IN THE SEGMENTS OF THE SPINAL CORD *(Continued)*

SEGMENT.	STRIPED MUSCLES.	REFLEX.	SKIN-FIELDS (CF. FIGS. 16 AND 17).
VIII C.	Pectoralis major (costal part). Pronator quadratus. Flexors of wrist and fingers. Latissimus. Radial lumbricales and interossei.	Palmar. Stroking palm causes closure of fingers.	Forearm and hand, inner half.
I T.	Lumbricales and interossei. Thenar and hypothenar eminences (C VII–T I).		Upper arm, inner half.
II to XII T.	Muscles of back and abdomen. Erectores spinæ (T I–LV). Intercostals (T I–T XII). Rectus abdominis (T V-T XII). External oblique (T V–XII). Internal oblique (T VII–L I). Transversalis (T VII–L I).	Epigastric. Tickling mammary region causes retraction of epigastrium (T IV–VII). Abdominal. Stroking side of abdomen causes retraction of belly (T IX–XII).	Skin of chest and abdomen in oblique dorso-ventral zones. The nipple lies between the zone of T IV and T V. The umbilicus lies in the field of T X.
I L.	Lower part of external and internal oblique and transversalis. Quadratus lumborum (L I–II). Cremaster. Psoas major and minor (?).	Cremasteric. Stroking inner thigh causes retraction of scrotum (L I–II).	Skin over lowest abdominal zone and groin.
II L.	Psoas major and minor. Iliacus. Pectineus. Sartorius (lower part). Flexors of knee (Remak). Adductor longus and brevis.		Front of thigh.
III L.	Sartorius (lower part). Adductors of thigh. Quadriceps femoris (L II-L IV). Inner rotators of thigh. Abductors of thigh.	Patellar tendon. Tapping tendon causes extension of leg. "Knee-jerk."	Front and inner side of thigh.
IV L.	Flexors of knee (Ferrier). Quadriceps femoris. Adductors of thigh. Abductors of thigh. Extensors of ankle (tibalis anticus). Glutei (medius and minor).	Gluteal. Stroking buttock causes dimpling in fold of buttock (L IV–V).	Mainly inner side of thigh and leg to ankle.
V L.	Flexors of knee (ham-string muscles) (L IV–S II). Outward rotators of thigh. Glutei. Flexors of ankle (gastrocnemius and soleus) (L IV–S II). Extensors of toes (L IV–S I). Peronæi.		Back of leg, and part of foot.

LOCALIZATION OF THE FUNCTIONS IN THE SEGMENTS OF THE SPINAL CORD (*Continued*)

SEGMENT.	STRIPED MUSCLES.	REFLEX.	SKIN-FIELDS (CF. FIGS. 16 AND 17).
I to II S.	Flexors of ankle (L V–S II). Long flexor of toes (L V–S II). Peronæi. Intrinsic muscles of foot.	Foot reflex. Extension of Achilles tendon causes flexion of ankle (S I–II). Ankle-clonus. Plantar. Tickling sole of foot causes flexion of toes or extension of great toe and flexion of others.	Back of thigh, leg, and foot; outer side.
III to V S.	Perineal muscles. Levator and sphincter ani (S I–III).	Vesical and anal reflexes.	Skin over sacrum and buttock. Anus. Perineum. Genitals.

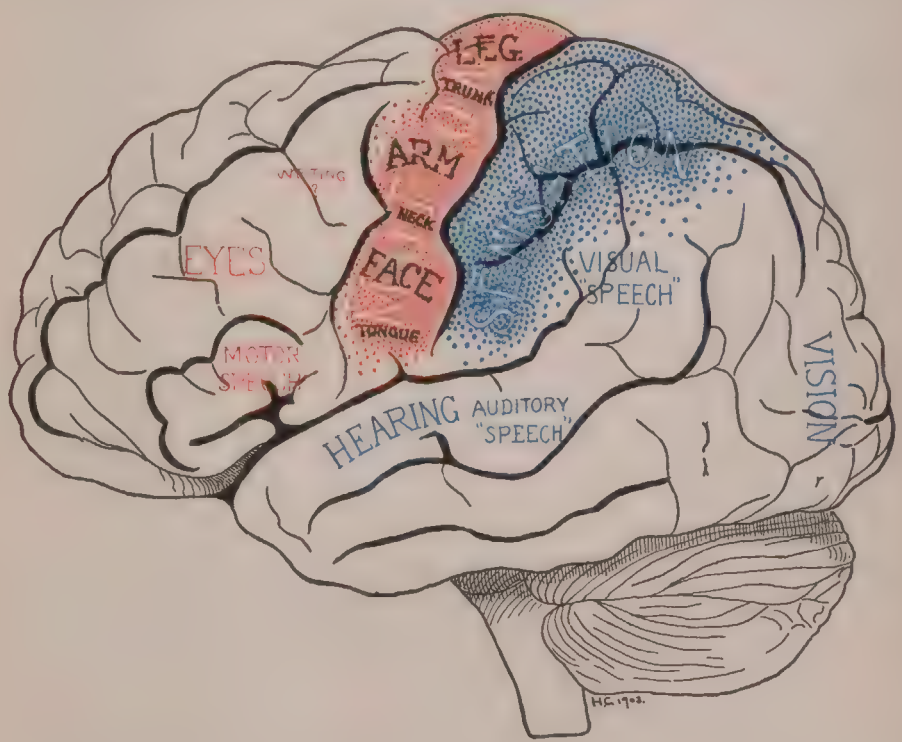


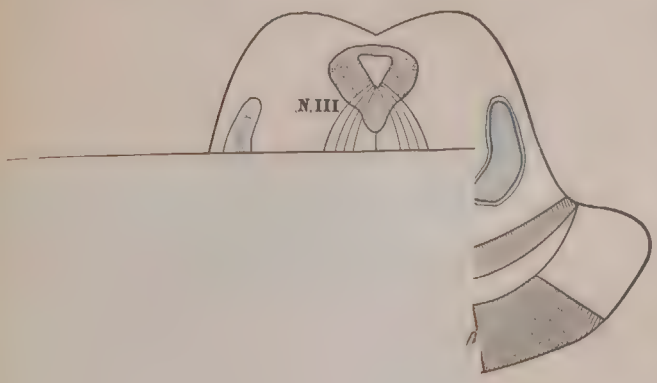
FIG. 12.—DIAGRAMMATIC REPRESENTATION OF CORTICAL LOCALIZATION IN THE LEFT HEMISPHERE, SHOWING THE SPEECH CENTRES. The motor areas determined by unipolar faradic excitation of the anthropoid cortex (Sherrington and Grünbaum) are here shown stippled in red and lie anterior to the Rolandic fissure. The sensory areas presumably lie posterior to this fissure and are roughly indicated in blue without accurate delineation. Lying as it does on the upper surface of the hemisphere, the leg area should not be visible on a lateral view such as is given here.

THE UPPER MOTOR SEGMENT AND MOTOR AREAS OF THE CORTEX.—The cell bodies of the upper motor neurones are found in the brain cortex lying for the most part in a strip anterior to the fissure of Rolando, and it is in this region that we find the movements of the body again represented.

The clinical studies of Hughlings Jackson, the experiments of Hitzig and Fritsch and of Ferrier, and the anatomical studies of tract myelinization by Flechsig laid the foundation for the great mass of most excellent work which has been done upon this subject. We owe much to Victor Horsley and his associates for their careful researches in this direction. True motor responses are elicited only by stimulation *anterior* to the Rolandic fissure; practically no point over the ascending frontal convolution fails to respond to stimulation. There is but slight extension of the motor cortex on to the paracentral lobule of the mesial surface of the brain. Movements are obtainable not only from the exposed part of the convolution, but also from its hidden surface to the very depths of the Rolandic sulcus. There is an area of representation for the trunk between the centres for the leg and arm, and also for the neck between those of the arm and face. The superior and inferior genua are the landmarks which indicate the situation of these small areas of representation

[illegible] of the medulla, after the fibres going to the cerebral nerves have crossed the middle line, a large proportion of the remaining fibres cross, decussating with those from the opposite pyramid, and pass into the opposite side of the spinal cord, forming the crossed pyramidal tract of the lateral column (fasciculus cerebrospinalis lateralis) (Fig. 15, 1). The smaller number of fibres which do not at this time cross descend in the ventral column of the same side, forming the

gray matter of the motor cortex pass into the white matter of the brain and form part of the corona radiata. They converge and pass between the basal ganglia in the internal capsule. Here the motor axis-cylinders are collected into a compact bundle—the pyramidal tract—occupying the knee and anterior two-thirds of the posterior limb of the internal capsule. The order in which the movements of the opposite side of the body are represented at this level, as learned from experimental observations on apes, is given in Fig. 13.



y Paths in Crura.

After passing through the internal capsule the fibres of the pyramidal tract leave the hemisphere by the crus, of which they occupy about the middle three-fifths (Fig. 14). The ps are represented nearest the middle line. he crus, some of its axis-cylinder processes to end in arborizations about the ganglion nerve on the opposite side; and in this way,

ird frontal or Broca's convolu- e is a separate centre presiding ne have assumed such a centre as indicated on the diagram. he opposite side has commonly external surface of the frontal be elicited from the occipital cortex, but probably none of these reactions are comparable to the more simple movements through the pyramidal tract which follow stimulation of the ascending frontal convolution.

The axis-cylinder processes of the upper motor neurones after leaving the

direct pyramidal tract, or Türck's column (fasciculus cerebro-spinalis ventralis) (Fig. 15, 2).

At every level of the spinal cord axis-cylinder processes leave the crossed pyramidal tract to enter the ventral horns and end about the cell bodies of the lower motor neurones. The tract diminishes in size from above downward. The fibres of the direct pyramidal tract cross at different levels in the ventral white commissure, and also, it is believed, end about cells in the ventral horns on the opposite side of the cord. This tract usually ends about the middle of the thoracic region of the cord.

The Sensory System.—The path for sensory conduction is more complicated than the motor path, and in its simplest form is composed of at least three sets of neurones, one above the other. The cell bodies of the lowest neurones are in the ganglia on the dorsal roots of the spinal nerves, and the ganglia of the sensory cerebral nerves. These ganglion cells have a special form, having apparently but a single process, which, soon after leaving the cell, divides in a T-shaped manner, one portion running into the central nervous system and the other to the periphery of the body. Embryological and comparative anatomical studies have made it seem probable that the peripheral sensory fibre, the process which conducts toward the cell, represents the protoplasmic processes, while that which conducts away from the cell is the axis-cylinder process. In the peripheral sensory nerves we have, then, the dendrites of the lower sensory neurones. These start in the periphery of the body from their various specialized end organs. The axis-cylinder processes leave the ganglia and enter the spinal cord by the dorsal roots of the spinal nerves. After entering the cord each axis-cylinder process divides into an ascending and a descending branch, which run in the dorsal fasciculi. The descending branch runs but a short distance, and ends in the gray matter of the same side of the cord. It gives off a number of collaterals, which also end in the gray matter. The ascending branch may end in the gray matter soon after entering, or it may run in the dorsal fasciculi as far as the medulla, to end about the nuclei there. In any case it does not cross the middle line. The lower sensory neurone is direct.

The cells about which the axis-cylinder processes and their collaterals of the lower sensory neurone end are of various kinds. They are known as sensory neurones of the second order. In the first place, some of them end about the cell bodies of the lower motor neurones, forming the path for reflexes. They also end about cells whose axis-cylinder processes cross the middle line and run to the opposite side of the brain. In the spinal cord these cells are found in the different parts of the gray matter, and their axis-cylinder processes run in the opposite ventro-lateral ascending tract of Gowers (Fig. 15, 6) and in the ground bundles (fasciculus lateralis proprius and fasciculus ventralis proprius).

In the medulla the nuclei of the dorsal fasciculi (nucleus fasciculi gracilis and nucleus fasciculi cuneati) contain for the most part cells of this character. Their axis-cylinder processes, after crossing, run toward the brain in the medial lemniscus or bundle of the fillet; certain of the longitudinal bundles in the formatio reticularis also represent sensory paths from the spinal cord and medulla toward higher centres. The fibres of the medial lemniscus or fillet do not, however, run directly to the cerebral cortex. They end about cells in

the ventro-lateral portion of the optic thalamus, and the tract is continued on by way of another set of neurones, which send processes to end in the cortex of the posterior central and parietal convolutions. This is the most direct path of sensory conduction, but by no means the only one. The peripheral sensory neurones may also end about cells in the cord whose axones run but a short distance toward the brain before ending again in the gray matter, and the path, if path it can be called, is made up of a series of these superimposed neurones. The gray matter of the cord itself is also believed to offer paths of sensory conduction. All these paths reach the tegmentum and optic thalamus, and thence are distributed to the cortex along with the other sensory paths. There may also be paths of sensory conduction through the cerebellum by way of the direct cerebellar tract and Gowers' bundle.

From this short summary it is evident that the possible paths for the conduction of afferent impulses are many, and become more complex as the various tracts approach the brain where our knowledge of them is somewhat indefinite. The anatomical arrangement of the two lower orders of sensory neurones is, however, sufficiently well understood to be of great clinical value. We have seen in the case of the motor neurones that the distribution of the peripheral nerves to the muscles, owing largely to the interlacing into plexuses of the neurones from the various spinal units, is quite different from that of the ventral roots themselves, and the same rule holds true for the peripheral nerve and dorsal root distribution for the cutaneous areas. The cutaneous fields corresponding to the peripheral nerves are well known, and although our knowledge of the exact site and outline of some of the segmental skin-fields, represented by the dorsal roots, is less accurately established, nevertheless they are sufficiently well understood to be of aid in determining the segmental level of spinal cord and of dorsal root lesions. Information concerning the topography in the adult of these skin units or dermatomes has been obtained from various sources; from morphological studies; from anatomical dissections; from physiological experimentation, particularly in Sherrington's hands; from the study of anæsthesias in clinical cases after traumatic injuries to the cord, and from Head's studies of the distribution of the cutaneous lesions in herpes zoster, and of the areas of referred pain and tenderness in visceral disease. The diagrams on pages 906 and 907 embody the results of many of these observations.

The cutaneous sensory impressions are in man conducted toward the brain, probably on the opposite side of the cord—that is, the path crosses to the opposite side soon after entering the cord. Muscular sense, on the other hand, is conducted on the same side of the cord in the fasciculus of Goll, to cross above by means of the axones of sensory neurones of the second order in the medulla.

Sensory Areas of the Brain.—Head and Holmes believe that there are two sensory centres—one in the optic thalamus, the other in a considerable area of the cerebral cortex. The thalamus plays a three-fold part in the fate of sensory impulses. Here all the afferent paths terminate; secondly, it contains a mass of gray matter which forms the centre for certain fundamental elements of sensation, particularly those capable of evoking pleasure and discomfort and consciousness of changes of state. Thirdly, in the lateral part of the thalamus is the centre through which the cortex influences the essential

thalamic centre, controlling and checking its activity. On their way from the periphery to the cortex afferent impulses pay toll to the co-ordinate mechanisms of the spinal cord and the cerebellum. At the thalamic junction they are re-grouped to act upon the two terminal centres. One of these, the essential organ of the optic thalamus, responds to all those elements which evoke consciousness of an internal change in state, more particularly pleasure and discomfort. Sensory impulses, then, pass by way of the internal capsule to the cortex, and these authors hold that in the main five groups of sensory impulses are distributed in this way: (1) those underlying postural recognition and the appreciation of passive movement and weight; (2) the impulses underlying the recognition of tactile differences; (3) those upon which depends the recognition of size and space; (4) those which enable us to localize the spot stimulated; and (5) thermal impulses.

These afferent materials are combined in the cortex with each other and with other sense impressions in intellectual processes. The cortical area concerned is that situated between the pre-central fissure and the occipital lobe.

The paths for the conduction of the stimuli which underlie the special senses are given in the section upon the cerebral nerves, and it is only necessary here to refer to what is known of the cortical representation of these senses.

Visual impressions are localized in the occipital lobes. The primary visual centre is on the mesial surface in the cuneus, especially about the calcarine fissure, and here are represented the opposite visual half-fields. Some authors believe that there is another higher centre on the outer surface of the occipital lobe, in which the vision of the opposite eye is chiefly represented. However this may be, most authors hold that the angular gyrus of the left hemisphere is a part of the brain in which are stored the memories of the meaning of letters, words, figures, and, indeed, of all seen objects. This is designated as the visual speech centre on the diagram (Fig. 12).

Auditory impressions are localized for the most part in the first temporal convolution and the transverse temporal gyri, and it is in this region in the left hemisphere that the memories of the meanings of heard words and sounds are stored. Musical memories are localized somewhat in front of those for words. The cortical centres for smell include a part of the base of the frontal lobe, the uncus, and perhaps the gyrus hippocampi. The centres for taste are supposed to be situated near those for smell, but we possess as yet no definite information about them.

Topical Diagnosis.—The successful diagnosis of the position of a lesion in the nervous system depends upon a careful and exhaustive examination into all the symptoms that are present, and then endeavoring with the help of anatomy and physiology to determine the place, a disturbance at which might produce these symptoms.

The abnormalities of motion are usually the most important localizing symptoms, both on account of the ease with which they can be demonstrated, and also because of the comparative accuracy of our knowledge of the motor path.

Lesions in any part of the motor path cause disturbances of motion. If destructive, the function of the part is abolished, and as the result there is *paralysis*. If, on the other hand, the lesion is an irritative one, the structures are thrown into abnormal activity, which produces *abnormal muscular con-*

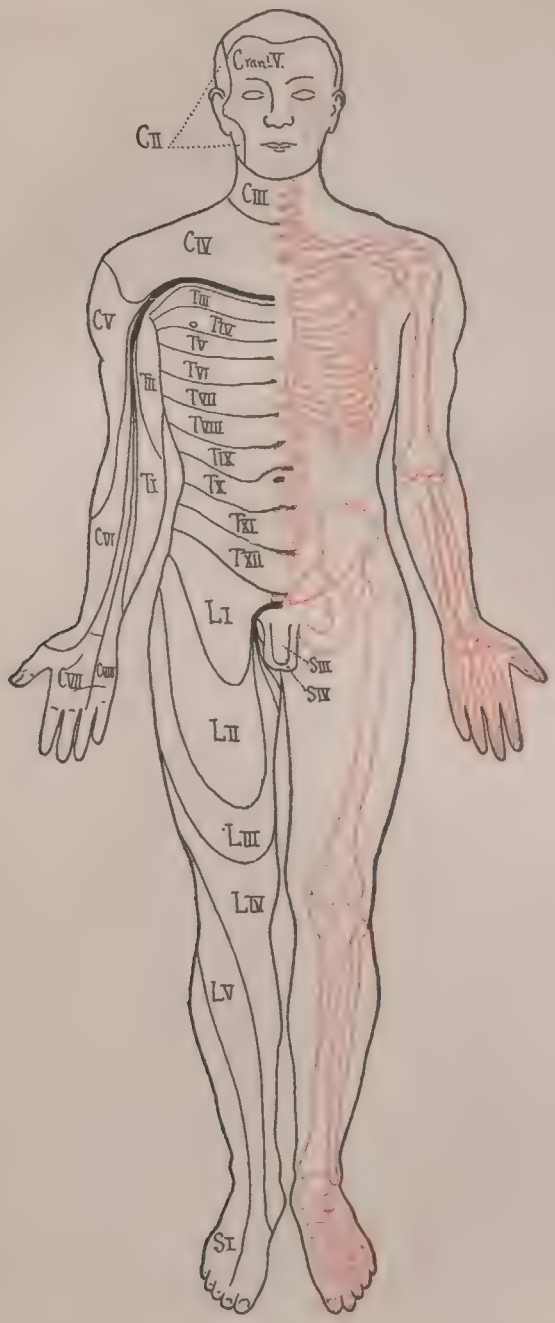


FIG. 16.—ANTERIOR ASPECT OF THE SEGMENTAL SKIN-FIELDS OF THE BODY, COMBINED FROM THE STUDIES OF HEAD, KOCHER, STARR, THORBURN, EDINGER, SHERRINGTON, WICHMANN, SEIFFER, BOLK, CUSHING, AND OTHERS. Heavy lines represent levels of fusior of dermatomes and the preaxial and postaxial lines of the limbs.

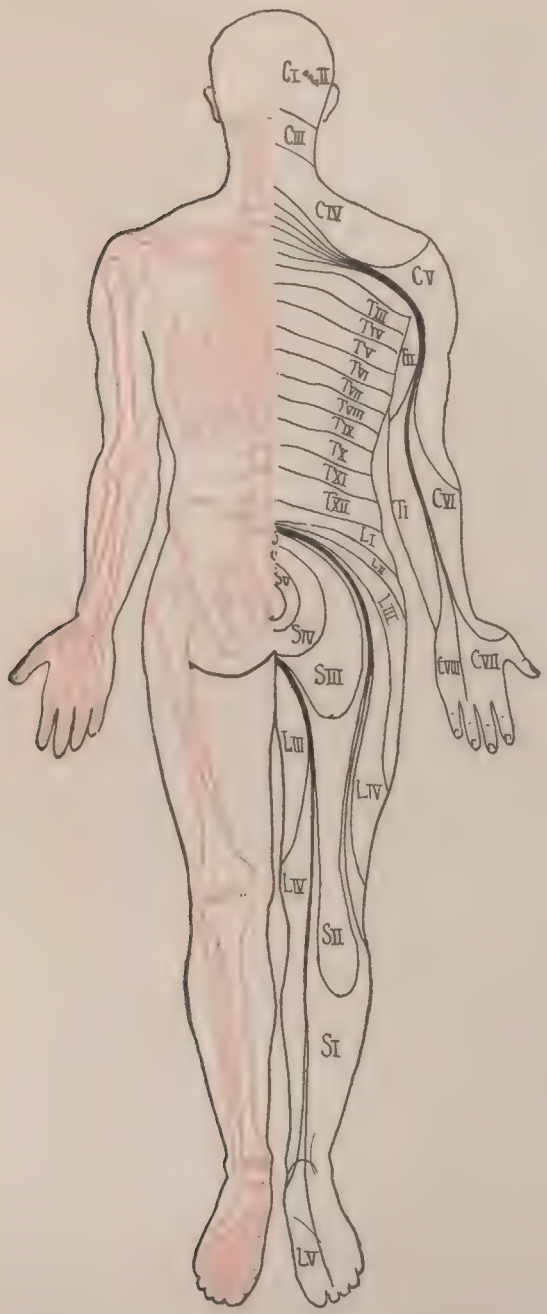


FIG. 17.—POSTERIOR ASPECT OF THE SEGMENTAL SKIN-FIELDS OF THE BODY.

traction. The character of the paralysis or of the abnormal muscular contraction varies with lesions of the upper and lower segment, the variations depending, first, upon the anatomical position of the two segments; and, secondly, upon the symptoms which are the result of secondary degeneration in each of the segments.

(*a*) Lesions of the Lower or Spino-muscular Segment.—*Destructive Lesions.*—It has been stated above that the nutrition of all parts of a neurone depends upon their connection with its healthy cell body; and if the cell body be injured, its processes undergo degeneration, or if a portion of a process be separated from the cell body, that part degenerates along its whole length. This so-called secondary degeneration plays a very important rôle in the symptomatology.

In the lower motor segment the degeneration not only affects the axis-cylinder processes which run in the peripheral nerves, but also the muscle fibres in which the axis-cylinder processes end. The degeneration of the nerves and muscles is made evident, first by the muscles becoming smaller and flabby, and, secondly, by change in their reaction to electrical stimulation. The degenerated nerve gives no response to either the galvanic or the faradic current, and the muscle does not respond to faradic stimulation, but reacts in a characteristic manner to the galvanic current. The contraction, instead of being sharp, quick, lightning-like, as in that of a normal muscle, is slow and lazy, and is often produced by a weaker current, and the anode-closing contraction may be greater than the cathode-closing contraction. This is the reaction of degeneration, but it is not always present in the classical form. The essential feature is the slow, lazy contraction of the muscle to the galvanic current, and when this is present the muscle is degenerated.

The myotatic irritability, or muscle reflex, and the muscle tonus depend upon the integrity of the reflex arc, of which the lower motor segment is the efferent limb, and in a paralysis due to lesion of this segment the muscle reflexes (tendon reflexes) are abolished and there is a diminished muscular tension.

Lower segment paralyses have for their characteristics degenerative atrophy with the reaction of degeneration in the affected muscles, loss of their reflex excitability, and a diminished muscular tension. These are the general characteristics, but the anatomical relations of this segment also give certain peculiarities in the distribution of the paralyses which help to distinguish them from those which follow lesions of the upper segment, and which also aid in determining the site of the lesion in the lower segment itself. The cell bodies of this segment are distributed in groups, from the level of the peduncles of the brain throughout the whole extent of the spinal cord to its termination opposite the second lumbar vertebra, and their axis-cylinder processes run in the peripheral nerves to every muscle in the body; so that the component parts are more or less widely separated from each other, and a local lesion causes paralysis of only a few muscles or groups of muscles, and not of a whole section of the body, as is the case where lesions affect the upper segment. The muscles which are paralyzed indicate whether the disease is in the peripheral nerves or spinal cord; for, as we have seen above, the muscles are represented differently in the peripheral nerves and in the spinal cord. Sensory symptoms, which may accompany the paralysis, are often of great

assistance in making a local diagnosis. Thus, in a paralysis with the characteristics of a lesion of the lower motor segment, if the paralyzed muscles are all supplied by one nerve, and the anæsthetic area of the skin is supplied by that nerve, it is evident that the lesion must be in the nerve itself. On the other hand, if the muscles paralyzed are not supplied by a single nerve, but are represented close together in the spinal cord, and the anæsthetic area corresponds to that section of the cord (see table), it is equally clear that the lesion must be in the cord itself or in its nerve roots.

Irritative Lesions of the Lower Motor Segment.—Lesions of this segment cause comparatively few symptoms of irritation. The fibrillary contractions which are so common in muscles undergoing degeneration are probably due to stimulation of the cell bodies in their slow degeneration, as in progressive muscular atrophy, or to irritation of the axis-cylinder processes in the peripheral nerves, as in neuritis. Lesions which affect the motor roots as they leave the central nervous system may cause spasmodic contractions in the muscles supplied by them. Certain convulsive paroxysms, of which laryngismus stridulus is a type, and to which the spasms of tetany also belong, are believed to be due to abnormal activity in the lower motor centres. These are the "lowest level fits" of Hughlings Jackson. Certain poisons, as strychnia and that of tetanus, act particularly upon these centres.

The lower motor segment may be involved in all diseases involving the peripheral nerves in cerebral and spinal meningitis, in injuries, in hæmorrhages and tumors of the medulla and cord or their membranes, in lesions of the gray matter of the segment, in anterior poliomyelitis, progressive muscular atrophy, bulbar paralysis, ophthalmoplegia, syringomyelia, etc.

(*b*) Lesions of the Upper Motor Segment.—*Destructive lesions* cause paralysis, as in the lower motor segment, and here again the secondary degeneration which follows the lesion gives to the paralysis its distinctive characteristics. In this case the paralysis is accompanied by a spastic condition, shown in an exaggeration of muscle reflex and an increase in the tension of the muscle. It is not accurately known how the degeneration of the pyramidal fibres causes this excess of the muscle reflex. The usual explanation is that under normal circumstances the upper motor centres are constantly exerting a restraining influence upon the activity of the lower centres, and that when the influence ceases to act, on account of disease of the pyramidal fibres, the lower centres take on increased activity, which is made manifest by an exaggeration of the muscle reflex.

We have seen that the neurones composing each segment of the motor path are to be considered as nutritional units, and therefore the secondary degeneration in the upper segment stops at the beginning of the lower. For this reason the muscles paralyzed from lesions in the upper segment do not undergo degenerative atrophy, nor do they show any marked change in their electrical reactions.

The separate parts of the upper motor segment lie much more closely together than do those of the lower segment, and therefore a small lesion may cause paralysis in many muscles. This is more particularly true in the internal capsule, where all the axis-cylinder processes of this segment are collected into a compact bundle—the pyramidal tract. A lesion in this region usually causes paralysis of most of the muscles on the opposite side of the

body—that is, hemiplegia. The pyramidal tract continues in a compact bundle, giving off fibres to the motor nuclei at different levels; a lesion anywhere in its course is followed by paralysis of all the muscles whose spinal centres are situated below the lesion. When the disease is above the decussation, the paralysis is on the opposite side of the body; when below, the paralyzed muscles are on the same side as the lesion. Above the internal capsule the path is somewhat more separated, and in the cortex the centres for the movements of the different sections of the body are comparatively far apart, and a sharply localized lesion in this region may cause a more limited paralysis, affecting a limb or a segment of a limb—the cerebral monoplegias; but even here the paralysis is not confined to an individual muscle or group of muscles, as is commonly the case in lower segment paralysis (see Fig. 10 and explanation).

To sum up, the paralyses due to lesions of the upper motor segment are widespread, often hemiplegic; the paralyzed muscles are spastic (the tendon reflexes exaggerated), they do not undergo degenerative atrophy, and they do not present the degenerative reaction to electrical stimulation.

There is an exception to the above statement—that is, in the paralyses which follow a complete transverse lesion of the spinal cord. Here the limbs are ~~of course~~ completely paralyzed, but instead of being spastic they are flaccid and the deep reflexes are absent. The muscles react normally to electricity. There is no satisfactory explanation of the loss of the reflexes under these conditions.

Irritative Lesions of the Upper Motor Segment.—Our knowledge of such lesions is confined for the most part to those acting on the motor cortex. The abnormal muscular contractions resulting from lesions so situated have as their type the localized convulsive seizures classed under Jacksonian or cortical epilepsy, which are characterized by the convulsion beginning in a single muscle or group of muscles and involving other muscles in a definite order, depending upon the position of their representation in the cortex. For instance, such a convulsion, beginning in the muscles of the face, next involves those of the arm and hand, and then the leg. The convulsion is usually accompanied by sensory phenomena and followed by a weakness of the muscles involved.

A majority of lesions of the motor cortex are both destructive and irritative—i. e., they destroy the nerve cells of a certain centre, and either in their growth or by their presence throw into abnormal activity those of the surrounding centres.

The upper motor segment is involved in nearly all the diseases of the brain and spinal cord, especially in injuries, tumors, abscesses, and hæmorrhages; transverse lesions of the cord; syringomyelia, progressive muscular atrophy, bulbar paralysis, etc. One lesion often involves both the upper and the lower motor segments, and there is paralysis in the different parts of the body, with the characteristics of each. Such a combination enables us in many cases to make an accurate local diagnosis.

Lesions in the optic path and in the different speech centres also give localizing symptoms, which should always be looked for.

(*c*) Lesions of the Sensory Path.—Here again the lesion may be either irritative or destructive. *Irritative lesions* cause abnormal subjective sensory impressions—paræsthesia, formication, a sense of cold or constriction, and pain of every grade of intensity. The character of the sensory symptoms

gives very little indication as to the position of the irritating process. Intense pain is, as a rule, a symptom of a lesion in the peripheral sensory neurones, but it may be caused by a disease of the sensory path within the central nervous system.

The exact distribution of symptoms gives more accurate data, for if they are confined to the distribution of a peripheral nerve or of a spinal segment the indication is plain. If one side of the body is more or less completely affected, the lesion is somewhere within the brain, etc.

Destructive Lesions.—A complete destruction of the sensory paths from any part of the body would of course deprive that part of sensation in all its qualities. This occurs most frequently from injury to the peripheral sensory neurones within the peripheral nerves, and the area of anæsthesia depends upon the nerve injured. Complete transverse lesion of the cord causes complete anæsthesia below the injury.

Unilateral lesions of the cord, medulla, dorsal part of the pons, tegmentum, thalamus, internal capsule, and cortex cause disturbances of sensation on the opposite side of the body; here again the extent of the defect more than its character helps us to determine the position of the lesion. Hemianæsthesia involving the face as well as the rest of the body can only occur above the place where the sensory paths from the fifth nerve have crossed the middle line on their way to the cortex. This is in the upper part of the pons. From this point to where they leave the internal capsule the sensory paths are in fairly close relation, and are at times involved in a very small lesion. Above the internal capsule the paths diverge quickly, and for this reason only an extensive lesion can involve them all, and in lesions of this part we are more apt to have the sensory disturbances confined to one or another region of the body. Unilateral lesions of the thalamus, pons, medulla, and cord usually cause sensory disturbances on the same side of the body, as well as those on the opposite side. These are due to the involvement of the sensory paths as they enter the central nervous system at or a little below the site of the lesion and before the axones of the sensory neurones of the second order have crossed the middle line. The area of disturbed sensation on the same side is limited to the distribution of one or more spinal segments and often indicates accurately the position and extent of the diseased process. As a rule, destructive lesions of the central nervous system do not involve all the paths of sensory conduction, and the loss of sensation is not complete. It is often astonishing how very slight the sensory disturbances are which result from an extensive lesion. Sensation may be diminished in all of its qualities, or, what is more common, certain qualities may be affected while others are normal. ~~These cases of dissociation of sensation, or so-called elective sensory paralysis, have been much studied of late.~~ Thus, the sense of pain and temperature may be lost while that of touch remains normal, as is often the case in diseases of the spinal cord, or there may be simply a loss of the muscular sense and of the stereognostic sense (the complex sensory impression which enables one to recognize an object placed in the hand), as occurs frequently from lesions of the cortex. Occasionally pain sensation persists with loss of tactile and thermic sensations. Almost every other combination has been described. It is the distribution more than the character of the sensory defect that is of importance, and often the distribution gives but uncertain indica-

tion of the position of the lesion. The combination of the sensory defect with different forms of paralysis gives the most certain diagnostic signs

B. SYSTEM DISEASES

I. INTRODUCTION

There are certain diseases of the nervous system which are confined, if not absolutely, still in great part, to definite tracts (combinations of neurones) which subserve like functions. These tracts are called *systems,* and a disease which is confined to one of them is a *system disease.* If more than one system is involved, the process is called a combined system disease. Just what diseases should be classed under these names has given rise to much discussion but to very little agreement. We can not speak positively; our knowledge is as yet not sufficiently accurate, either in regard to the exact limits of the systems themselves, or to the nature and extent of the morbid process in the several diseases.

It may be said that the nervous system is composed of two great systems of neurones, the afferent or sensory system and the efferent or motor system, and the connections between them. (See General Introduction.)

Locomotor ataxia is a disease confined at its onset to the afferent system, and progressive muscular atrophy is one of the efferent system. Representing typical system diseases as we now understand them, they have been taken as the basis of the classification. Several theories have been advanced to explain why a disease should be limited to a definite system of neurones. One view is based upon the idea that in certain individuals one or the other of these systems has an innate tendency to undergo degeneration; another assumes that neurones with a similar function have a similar chemical construction (which differs from that of neurones with a different function), and this is taken to explain why a poison circulating in the blood should show a selective action for a single functional system of neurones.

In the afferent tract locomotor ataxia stands alone as a system disease, and we now believe that herpes zoster is an inflammation of the dorsal root ganglia and stands in the same relation to tabes that acute anterior poliomyelitis does to chronic progressive muscular atrophy. In the efferent tract progressive (central) muscular atrophy is the chief representative, as in it the whole motor path is more or less involved. Theoretically, primary lateral sclerosis is a disease confined to the upper segment of the efferent tract, while chronic anterior poliomyelitis involves the lower segment of the tract.

In connection with locomotor ataxia, general paralysis is considered on account of their frequent association and as they are but different expressions of one and the same morbid process; and with progressive (central) muscular atrophy, the other forms of muscular atrophy are considered as a matter of convenience. In other instances, too, diseases are arranged in positions to which they might not be entitled, had a rigid classification of system diseases been maintained.

Spinal Fluid Compression Syndrome = Hitchcock.
J.A.M.A. May 19, 17.

results from compression of the cord.

1. Normal persons
2. Marked increase in proteins
3. Spontaneous coagulation = "massive"
4. Cells not increased.
5. Xanthochromia. (yellow color)

cf. agricultural machinery Stat. Sess. 1917-18 (other items of)
hereditary unemployment credits - Shiraz expenditure.

II. DISEASES OF THE AFFERENT OR SENSORY SYSTEM

1. LOCOMOTOR ATAXIA

(*Tabes Dorsalis; Posterior Spinal Sclerosis*)

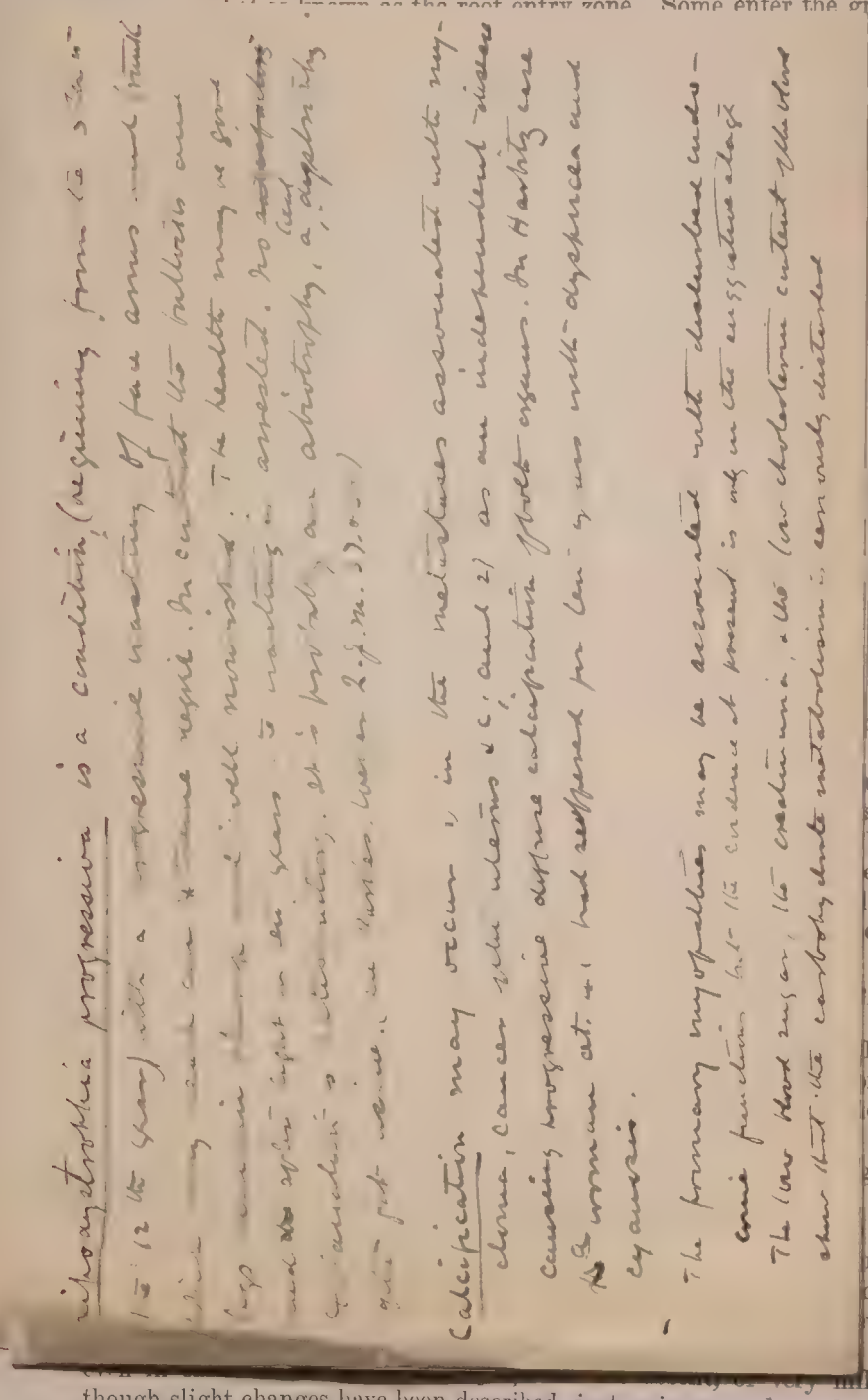
Definition.—An affection characterized clinically by sensory disturbances, incoördination, trophic changes, and involvement of the special senses, particularly the eyes. Anatomically there are found degenerations of the root fibres of the dorsal columns of the cord, of the dorsal roots, and at times of the spinal ganglia and peripheral nerves. Degenerations have been described in the brain, particularly the cortex cerebri, in the ganglion cells of the cord, and in the endogenous fibres of the dorsal columns.

Etiology.—It is a widespread disease, more frequent in cities than in the country. The relative proportion may be judged from the fact that of 16,562 cases in the neurological dispensary of the Johns Hopkins Hospital there were 201 cases of locomotor ataxia. Males are attacked more frequently than females, the proportion being nearly 10 to 1. The disease, although uncommon in the negro, is seen in them more frequently than some authors state. It is a disease of adult life, the great majority of cases occurring between the thirtieth and fiftieth years. Occasionally cases are seen in young men, and it may occur in children with hereditary syphilis. Syphilis is the important cause. In the Johns Hopkins Hospital the percentage, as found by Thomas, was 63.1. Erb's figures are most striking—of 300 cases of tabes in private practice, 89 per cent. had had syphilis. Moebius goes so far as to say, "The longer I reflect upon it, the more firmly I believe that tabes never originates without syphilis," and recent results of cases tested with the Wassermann reaction bear out this statement.

Contributing causes are excessive fatigue, overexertion, injury, exposure to cold and wet, and sexual excesses. There are instances in which the disease has closely followed severe exposure. Trauma has been noted in a few cases. Alcoholic excess does not seem to predispose to the disease. There are now a good many cases on record of the existence of the disease in both husband and wife, and a few in which the children were also affected.

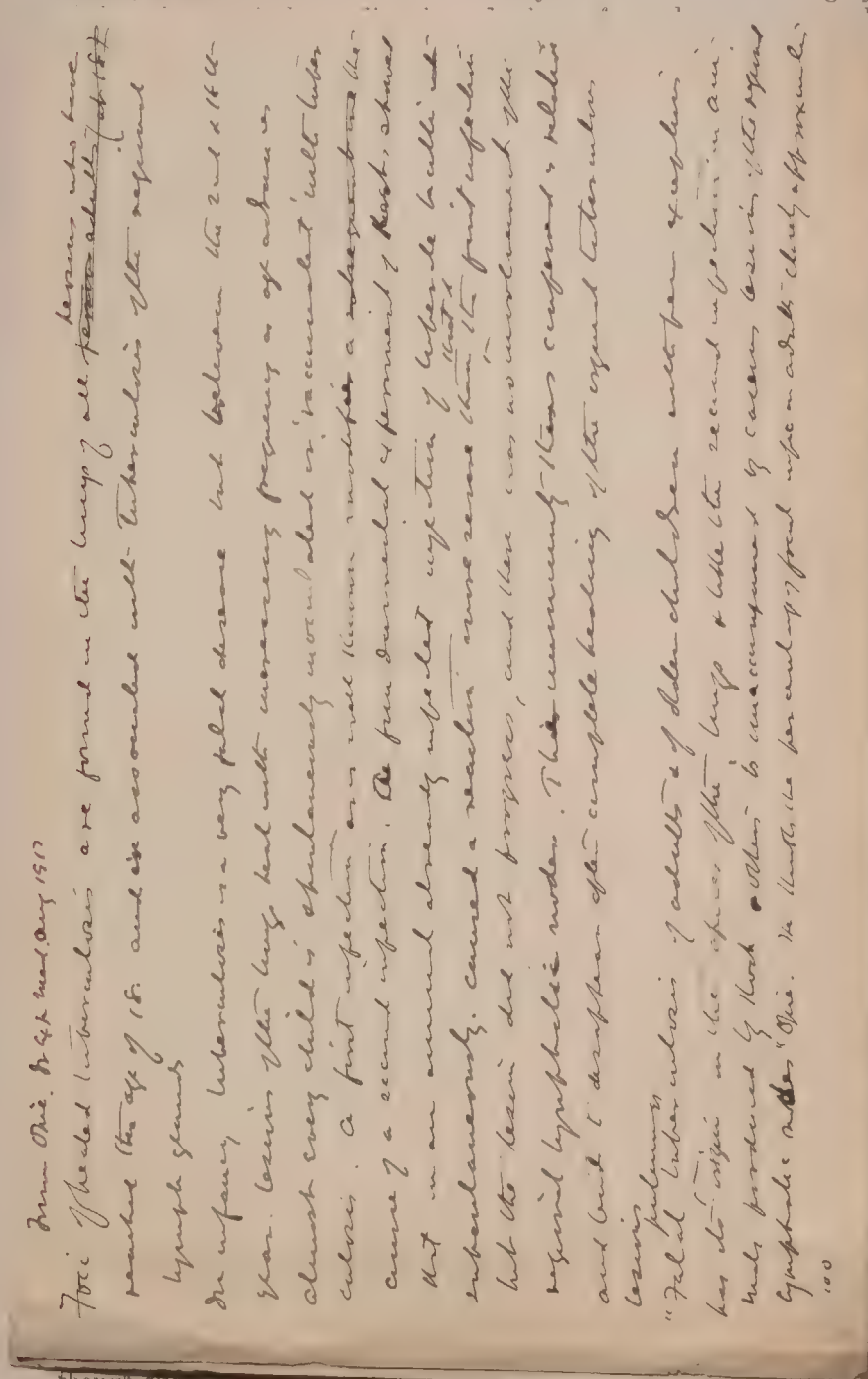
Morbid Anatomy and Pathology.—Posterior spinal sclerosis, although the most obvious gross change, is now no longer, as in Romberg's time, an adequate description of the condition. The dorsal fibres are of two kinds, those with their cell bodies outside the cord in the spinal ganglia, the so-called exogenous, or root fibres, and those which arise from cells within the cord, the endogenous fibres. These two sets occupy fairly well-determined regions, and a study of early cases of tabes has shown that it is the exogenous or root fibres that are first affected. The fibres of the dorsal roots enter the cord in two divisions, an external and an internal; the former is composed of fibres of small calibre, which, in the cord, make up Lissauer's tract, and occupy the space between the apex of the dorsal cornua and the periphery of the cord, and really do not form part of the dorsal columns. They are short, soon entering the gray matter, and do not seem to be affected, or only slightly so, in early cases.

The larger fibres enter the cord by the internal division, just medial to the



though slight changes have been described, just as in general paralysis, without marked tabetic symptoms, there may be degeneration of the dorsal columns.

cornua, in what is known as the root entry zone. Some enter the gray matter



marked tabetic symptoms, there may be degeneration of the dorsal columns.

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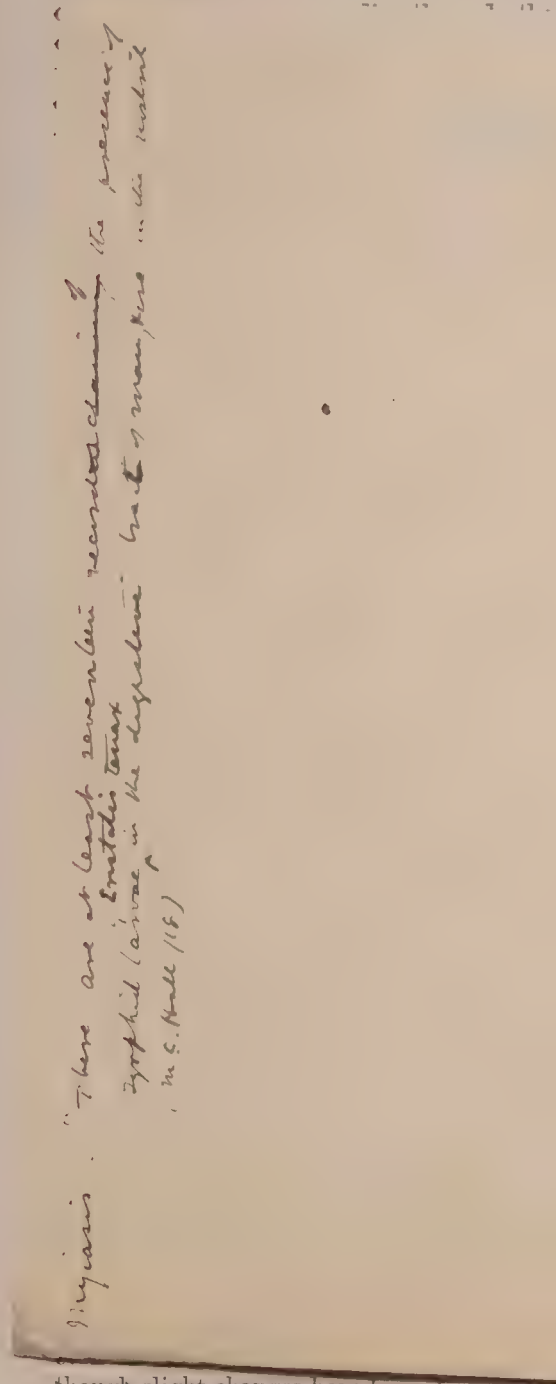

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cornua, in what is known as the root entry zone. Some enter the gray matter of the spinal cord almost directly and others after a longer course, while still others run in the cord to the medulla, to end in the nuclei of the dorsal columns. As the fibres of every spinal nerve enter the cord between the dorsal cornua and the nerve fibres which have entered lower down, the fibres from each root are successively pushed more and more toward the median line, and so in the cervical cord the fasciculi of Goll are largely composed of long fibres derived from the sacral and lumbar roots.

That it is the coarse dorsal root fibres which are first affected in tabes is generally admitted, but there is much divergence of opinion as to the character and location of the initial process.

Nageotte calls attention to the frequency of a transverse, interstitial neuritis of the dorsal roots just after they have left the ganglia and are still surrounded by the dura, and he believes that it is this neuritis which is the primary lesion, ~~in tabes~~. Obersteiner and Redlich have laid great stress on the presence of an inflammation of the pia mater over the dorsal aspect of the cord, which involves the root fibres as they pass through. They point out that it is just here that the dorsal roots are most vulnerable, for at this point —that is, while surrounded by the pia—they are almost completely devoid of their myelin sheaths. Changes in the blood-vessels of the cord, of the pia, and of the nerve roots have been described in early tabes, and Marie and Guillain ~~have~~ advanced the belief that the changes in the cord are due to an affection (syphilis) of the posterior lymphatic system ~~which is~~ confined to the dorsal columns of the cord, the pia mater over them, and the dorsal roots. For them the changes in the nervous system are only apparently radicular or systemic.

With the Marchi stain, degeneration of the root fibres in the root-entry zone is a constant finding. This change is radicular in the sense that it varies in intensity with the different roots and is most marked in the sacral and lumbar regions. The degeneration is not found in the dorsal roots, but begins within the cord just beyond where the root fibres lose their neurolemma and their myelin sheaths. Degenerated fibres may be traced into the dorsal gray matter and among the ganglion cells of the columns of Clarke. The long columns which ascend the cord also degenerate.

In more advanced cases, in addition to the lesion described above, there are degeneration of the dorsal roots and some alteration of the cells in the spinal ganglia. The fibres distal to the ganglia are practically normal, although at times the sensory fibres, at the periphery of a limb, show degeneration. Within the cord, the exogenous fibres are diseased as already described; there is also degeneration in the endogenous system of fibres. Optic atrophy is frequently found. The other cranial nerves, especially the fifth with its ganglion, have been found degenerated.

The disease occasionally spreads beyond the sensory system in the cord, and in advanced cases the cells in the ventral horns may be degenerated in association with muscular atrophy. Mott very generally found more or less marked changes in the pyramidal fibres; these he believed to be evidence of changes in the cerebral cortex. Degeneration of the cortex may exist, but even in cases where the mental symptoms are absent, or very mild, similar though slight changes have been described, just as in general paralysis, without marked tabetic symptoms, there may be degeneration of the dorsal columns.

Pupil symptoms common to syphilis + paresis – meningo-syphilis.

Progressive nerve diseases sometimes.

Paralysis of vocal cords often – laryngeal muscles paralyzed or paretic.

The close association, or even identity, of tabes and general paralysis will be considered later.

Symptoms.—These are best considered under three stages—the incipient stage, the ataxic stage, and the paralytic stage.

INCIPIENT STAGE.—This is sometimes called the pre-ataxic stage. The manner in which tabes makes its onset differs very widely in the different cases, and mistakes in diagnosis are often made early in the disease. The following are the most characteristic initial symptoms:

Pains, usually of a sharp stabbing character; hence, the term lightning pains. They last for only a second or two and are most common in the legs or about the trunk, and tend to follow dorsal root areas. They dart from place to place. At times they are associated with a hot burning feeling and often leave the affected area painful to pressure, and occasionally herpes may follow. The intensity of the pain varies from a sore, burning feeling of the skin to a pain so intense that, were it not for momentary duration, it would exceed human endurance. They occur at irregular intervals, and are prone to follow excesses or to come on when health is impaired. When typical, these pains are practically pathognomonic of the condition. The gastric crises and other crises may occur. Paræsthesia may also be among the first symptoms—numbness of the feet, tingling, etc.—and at times a sense of constriction about the body.

Ocular Symptoms.—(*a*) Optic atrophy. This occurs in about 10 per cent. of the cases, and is often an early and even the first symptom. There is a gradual loss of vision, which in a large majority of cases leads to total blindness. (*b*) Ptosis, which may be double or single. (*c*) Paralysis of the external muscles of the eye. This may be of a single muscle or occasionally of all the muscles of the eye. The paralysis is often transient, the patient merely complaining that he saw double for a certain period. (*d*) Argyll-Robertson pupil, in which there is loss of the iris reflex to light but contraction during accommodation. The pupils are often very small—spinal myosis.

Bladder Symptoms.—The first warning of the disease which the patient has may be a certain difficulty in emptying the bladder. Incontinence of urine occurs only at a later stage of the disease. Decrease in sexual desire and power may also be an early symptom.

Trophic Disturbances.—These usually occur later in the disease, but at times they are very early symptoms, and it is not very infrequent to have one's attention called to the trouble by the presence of a perforating ulcer or of a characteristic Charcot's joint.

Loss of the Deep Reflexes.—This early and most important symptom may occur years before the development of ataxia. Even alone it is of great moment, since it is very rare to meet with individuals in whom the knee and ankle jerks are normally absent. The combination of loss of either of these with one or more of the symptoms mentioned above, especially with the lightning pains and ptosis or Argyll-Robertson pupil, is practically diagnostic. These reflexes gradually decrease, and one may be lost before the other, or disappear first in one leg.

These are the most common symptoms of the initial stage of tabes and may persist for years without the development of incoördination. The patient may look well and feel well, and be troubled only by occasional attacks of

lightning pains or of one of the other subjective symptoms. Moebius goes so far as to state that the typical Argyll-Robertson pupil means either tabes or general paralysis, and that paralysis of the external muscles of the eye developing in adults is of almost equal importance, especially if it develops painlessly.

The time between the syphilitic infection and the occurrence of the first symptoms of locomotor ataxia varies within wide limits. About one-half the cases occur between the sixth and fifteenth year, but many begin even later than this.

The disease may never progress beyond this stage, and when optic atrophy develops early and leads to blindness, ataxia rarely, if ever, supervenes, but the mental symptoms of paresis not infrequently follow, a sequence which must be kept in mind. There is a sort of antagonism between the ocular symptoms and the progress of the ataxia. Charcot laid considerable stress upon this, and both Dejerine and Spiller have since emphasized the point.

Ataxic Stage.—*Motor Symptoms.*—The ataxia is believed to be due to a disturbance or loss of the afferent impulses from the muscles, joints, and deep tissues, and a disturbance of the muscle sense itself can usually be demonstrated. It develops gradually. One of the first indications to the patient is inability to get about readily in the dark or to maintain his equilibrium when washing his face with the eyes shut. When the patient stands with the feet together and the eyes closed, he sways and has difficulty in maintaining his position (Romberg's symptom), and he may be quite unable to stand on one leg. He does not start off promptly at the word of command. On turning quickly he is apt to fall. He descends stairs with more difficulty than he ascends them. Gradually the characteristic ataxic gait develops. The patient, as a rule, walks with a stick, the eyes are directed to the ground, the body is thrown forward, and the legs are wide apart. In walking, the leg is thrown out violently, the foot is raised too high and is brought down in a stamping manner with the heel first, or the whole sole comes in contact with the ground. Ultimately the patient may be unable to walk without the assistance of two canes. This gait is very characteristic, and unlike that seen in any other disease. The incoördination is not only in walking, but in the performance of other movements. If the patient is asked, when in the recumbent posture, to touch one knee with the other foot, the irregularity of the movement is very evident. Incoördination of the arms is less common, but usually develops in some grade. It may in rare instances exist before the incoördination of the legs. It may be tested by asking the patient to close his eyes and to touch the tip of the nose or the tip of the ear with the finger, or with the arms thrust out to bring the tips of the fingers together. The incoördination may early be noticed by a difficulty which the patient experiences in buttoning his collar or in performing one of the ordinary routine acts of dressing.

One of the most striking features of the disease is that with marked incoordination there is but little loss of muscular power. The grip of the hands may be strong and firm, the power of the legs, tested by trying to flex them, may be unimpaired, and their nutrition, except toward the close, may be unaffected.

There is a remarkable muscular relaxation (hypotonia) which enables the

anaesthesia of hand, penis. impotence.

hyperaesthetic or anaesthetic zones. must often in midst of leg often midst of area

also as girdle sensation girdle abnormality. may be insulated areas early.

may affect equally tactile, pain or temp. sense. Pain often to most affected

afterwards. finally localization.

Loss of deep sensibility + movement sense. also anaesthesia of deep tissues viscera

Radicular irritative phenomena. = Sensory. abnormality. Vaso. ex. Ans. constant.

Partial crises. Loss thus sporadic contract. of stem. "tingles"

ala ocular crises. [illegible]

Paresthesia. early. often ones affected by pain.

joints to be placed in positions of hyperextension and hyperflexion. It gives sometimes a marked backward curve to the legs.

Sensory Symptoms.—The lightning pains may persist. They vary greatly in different cases. Some patients are rendered miserable by the frequent occurrence of the attacks; others escape altogether. In addition, common symptoms are tingling, pins and needles, particularly in the feet, and areas of hyperæsthesia or of anæthesia. The patient may complain of a change in the sensation in the soles of the feet, as if cotton was interposed between the floor and the skin. Sensory disturbances occur less frequently in the hands. Objective sensory disturbances can usually be demonstrated, and, indeed, almost every variety of sensory disturbance has been described. Bands of a moderate grade of anæsthesia about the chest are not uncommon; they are apt to follow the distribution of spinal segments. The most marked disturbances are usually found on the legs. Retardation of the sense of pain is common, and a pin prick on the foot is first felt as a simple tactile impression, and the sense of pain is not perceived for a second or two or may be delayed for as much as ten seconds. The pain felt may persist. A curious phenomenon is the loss of the power of localizing the pain. For instance, if the patient is pricked on one limb he may say that he feels it on the other (allocheiria), or a pin prick on the foot may be felt on both feet. The muscular sense which is usually affected early, becomes much impaired and the patient no longer recognizes the position in which his limbs are placed. This may be present in the pre-ataxic stage.

Reflexes.—As mentioned, the loss of the knee and ankle jerks is one of the earliest symptoms of the disease. Occasionally a case is found in which they are retained, and anatomically it has been shown that in these cases the lumbar segments were little if at all involved. The skin reflexes may at first be increased, but later are usually involved with the deep reflexes.

Special Senses.—The eye symptoms noted above may be present, but, as mentioned, ataxia is rare with atrophy of the optic nerve.

Deafness may occur, due to lesion of the auditory nerve. There may also be attacks of vertigo. Olfactory symptoms are rare.

Visceral Symptoms.—Among the most remarkable sensory disturbances are the tabetic crises, severe paroxysms of pain referred to various viscera; thus, laryngeal, gastric, nephric, rectal, urethral, and clitoral crises have been described. The most common are the gastric and laryngeal. Gastric crises may occur early and persist as the most prominent feature. Starr found them as the first symptom 18 times in 450 cases. The onset is usually sudden, with pain of a severe burning, twisting type in the epigastrium, radiating to the back and behind the sternum. Vomiting follows the pain, and may be quite independent of food. Pallor, sweating, cold extremities, and a small pulse are associated, and in rare instances death occurs in collapse. The attacks are not unlike and are probably of the same nature as the so-called abdominal angina pectoris. The blood pressure may be very high, as reported by Barker, and it seems not improbable that the condition is associated with angiospasm in the territory of the gastric and mesenteric vessels. No special change may be found at autopsy. In the laryngeal crises there may be true spasm with dyspnœa and noisy inspiration. A patient may die in the attack. There are also nasal crises, associated with sneezing fits.

The sphincters are frequently involved. Early in the disease there may be a retardation or hesitancy in making water. Later there is retention, and cystitis may occur. Unless great care is taken the inflammation may extend to the kidneys. Constipation is extremely common. Later in the disease the sphincter ani is weakened. The sexual power is usually lost in the ataxic stage.

Trophic Changes.—Skin rashes, such as herpes, œdema, or local sweating, may develop in the course of the lightning pains. Alteration in the nails may occur. A perforating ulcer may develop on the foot, usually beneath the great toe. A perforating buccal ulcer has also been described. Onychia may prove very troublesome.

Arthropathies (Charcot's Joints).—Anatomically there are: (1) enlargement of the capsule with thickening of the synovial membranes and increase in the fluids; (2) slight enlargement of the ends of the bones, with slight exostoses; (3) a dull velvety appearance of the cartilages, with atrophy in places. (V. E. Henderson). The knees are most frequently involved. The spine is affected in rare instances. Recurring trauma is an important element in the causation, but trophic disturbances have a strong influence in the etiology. A striking feature is the absence of pain. Suppuration may occur, also spontaneous fractures. Among other trophic disturbances may be mentioned atrophy of the muscles, which is usually a late manifestation, but may be localized and associated with neuritis. In any very large collection of cases many instances of atrophy are found, due either to involvement of the ventral horns or to peripheral neuritis.

Aneurism is found in many cases, in as high as 20 per cent. of some series.

Cerebral Symptoms.—Hemiplegia may develop at any stage of the disease, more commonly when it is well advanced. It may be due to hæmorrhagic softening in consequence of disease of the vessels or to progressive cortical changes. Hemianæsthesia is sometimes present. Very rarely the hemiplegia is due to coarse syphilitic disease.

Dementia paralytica frequently exists with tabes; indeed, we have come to regard these two diseases as simply different localizations of the same morbid process. In other instances melancholia, dementia, or paranoia occur.

Paralytic Stage.—After persisting for an indefinite number of years the patient gradually loses the power of walking and becomes bedridden or paralyzed. In this condition he is very likely to be carried off by some intercurrent affection, such as pyelo-nephritis, pneumonia, or tuberculosis.

The Course of the Disease.—A patient may remain in the pre-ataxic stage for an indefinite period; and the loss of knee-jerk and the gray atrophy of the optic nerves may be the sole indication of the true nature of the disease. In such cases incoördination rarely develops. In a majority of cases the progress is slow, and after six or eight years, sometimes less, the ataxia is well developed. The symptoms may vary a good deal; thus, the pains, which may have been excessive at first, often lessen. The disease may remain stationary for years; then exacerbations occur and it makes rapid progress. Occasionally the process seems to be arrested. There are instances of what may be called acute ataxia, in which, within a year or even less, the incoördination is marked, and the paralytic stage may develop within a few months. The disease itself

Spembancem fractelli - Morgha fracilem

und antic nicoffeinen?

blood Wassermann. 70%

Sp. fluid. " - 60-80%

positive globulin. 50-55%

lymphocytosis in sp. fluid - in 50%

[illegible]

colloidal gold test - [illegible]

[illegible]

rarely causes death, and after becoming bedridden the patient may live for fifteen or twenty years.

Diagnosis.—In the initial stage the lightning pains are almost distinctive, and when combined with any of the other signs are quite so. The association of progressive atrophy of the optic nerves with loss of knee-jerk is also characteristic. The early ocular palsies are of the greatest importance. A squint, ptosis, or the Argyll-Robertson pupil may be the first symptom, and may exist with the loss only of the knee-jerk. Loss of the knee-jerk alone, however, does occasionally occur in healthy individuals. A history of preceding syphilis lends added weight to the symptoms, and its presence or absence may be of the utmost importance in determining the diagnosis. The Wassermann reaction is present in a large proportion of all cases, and a study of the spinal fluid may be a help in doubtful cases (see General Paresis).

The diseases most likely to be confounded with locomotor ataxia are: (*a*) Peripheral Neuritis.—The steppage gait of arsenical, alcoholic, or diabetic paralysis is quite unlike that of locomotor ataxia. In these forms there is a paralysis of the feet, and the leg is lifted high in order that the toes may clear the floor. The use of the word *ataxia* in this connection should no longer be continued. In the rare cases in which the muscle sense nerves are particularly affected and in which there is true ataxia, the absence of the lightning pains and eye symptoms and the history will suffice in a majority of cases to make the diagnosis clear. In diphtheritic paralysis the early loss of the knee-jerk and the associated eye symptoms may suggest tabes, but the history, the existence of paralysis of the throat, and the absence of pains render a diagnosis easy.

(*b*) Ataxic Paraplegia.—Marked incoördination with spastic paralysis is characteristic of the condition which Gowers has termed ataxic paraplegia. In a majority of the cases this affection is distinguished also by the absence of pains and of eye symptoms, but it may be a manifestation of the cord lesions in tabo-paralysis.

(*c*) Cerebral Disease.—In diseases of the brain involving the afferent tracts ataxia is at times a prominent symptom. It is usually unilateral or limited to one limb; this, with the history and the associated symptoms, excludes tabes.

(*d*) Cerebellar Disease.—The cerebellar incoördination has only a superficial resemblance to that of locomotor ataxia, and is more a disturbance of equilibrium than a true ataxia; the knee-jerk is usually present, there are no lightning pains, no sensory disturbances; while, on the other hand, there are headache, optic neuritis, and vomiting.

(*e*) Acute syphilitic affections involving the dorsal columns of the cord may be associated with incoördination and resemble tabes very closely. In a case under my care, the gait was characteristic and Romberg's symptom was present. The knee-jerk, however, was retained and there were no ocular symptoms. The condition had developed within three months, and there was a well-marked history of syphilis. Under large doses of iodide of potassium the ataxia and other symptoms completely disappeared.

(*f*) General Paresis.—Even though these two diseases are so nearly allied and often associated, it is of very great practical importance to determine, when possible, whether the type is to be spinal or cerebral, for, in the great majority of cases, when this is established, it does not change. The

difficulty arises in the premonitory stage, when ocular changes and abnormalities of sensation and the deep reflexes may be the only symptoms. At this stage any alteration in the mental characteristics is of the utmost significance. (See General Paresis.) Loss of the deep reflexes and lightning pains speak for tabes; active reflexes, with ocular changes, especially optic atrophy, are suggestive of paresis.

(*g*) VISCERAL CRISES and NEURALGIC SYMPTOMS may lead to error, and in middle-aged men with severe, recurring attacks of gastralgia it is always well to bear in mind the possibility of tabes, and to make a careful examination of the eyes and of the knee-jerk.

Prognosis.—Complete recovery can not be expected, but arrest of the process is not uncommon and a marked amelioration of the symptoms is frequent. Optic-nerve atrophy, one of the most serious events in the disease, has this hopeful aspect—that incoördination rarely follows and the progress of the spinal symptoms may be arrested. On the other hand, mental symptoms are more likely to follow. The optic atrophy itself is occasionally checked. On the whole, the prognosis in tabes is bad. The experience of such men as Weir Mitchell, Charcot, and Gowers is distinctly opposed to the belief that locomotor ataxia is ever completely cured. There is more hope now that in very early cases coming on soon after infection the course may be arrested by salvarsan. Death is usually from some cardio-vascular complication; next in frequency from tuberculosis and pneumonia (Burr).

Treatment.—To arrest the progress and to relieve, if possible, the symptoms are the objects which the practitioner should have in view. A quiet, well-regulated method of life is essential. It is not well, as a rule, for a patient to give up his occupation so long as he is able to keep about and perform ordinary work, provided there is no evident mental change. I know tabetics who have for years conducted large businesses, and there have been several notable instances in our profession of men who have risen to distinction in spite of the existence of this disease. Excesses of all sorts, more particularly *in baccho et venere,* should be carefully avoided. A man in the pre-ataxic stage should not marry.

Care should be taken in the diet, particularly if gastric crises have occurred. To secure arrest of the disease many remedies have been employed. Salvarsan in small repeated doses (0.2 gm.) should be tried in early cases, though the published results have not, on the whole, been satisfactory. Neither mercury nor the iodide of potassium has anything like the same influence over the tabetic lesions that they have over the ordinary syphilitic processes. However, when the syphilis is comparatively recent, when symptoms develop within two years of the primary infection, the disease may be arrested by mercury and iodide of potassium. The French authors have recently spoken much more hopefully of the benefit of anti-syphilitic treatment in early cases of tabes, and it is well to give the patient the benefit of salvarsan and a thorough course of mercurial inunctions and iodide of potassium. Of remedies which may be tried and are believed by some writers to retard the progress, the following are recommended: Arsenic in full doses, nitrate of silver in quarter-grain doses, Calabar bean, ergot, and the preparations of gold.

For the pains, complete rest in bed, as advised by Weir Mitchell, and counter-irritation to the spine (either blisters or the thermo-cautery) may

Tubes superior - progressive worse by vision & bulbar symptoms.

average duration - 10-20 years.

Deutsch-Amerikanische Lichtbildgesellschaft m.b.H. Berlin SW 68, Lindenstr. 3.

General Hydrotherapy in Dr. Dowdell's Sanatorium

be employed. The severe spells which come on particularly after excesses of any kind are often promptly relieved by a hot bath or by a Turkish bath. For the severe recurring attacks of lightning pains spinal cocainization may be tried. A prolonged course of nitrate of silver seems in some cases to allay the pains and lessen the liability to the attacks. Antipyrin and antifebrin may be employed, and occasionally do good, but their analgesic powers in this disease have been greatly overrated. Cannabis indica is sometimes useful. In the severe paroxysms of pain hypodermics of morphia or of cocaine must be used. The use of morphia should be postponed as long as possible. Electricity is of very little benefit. For the severe attacks of gastralgia morphia is also required. Gastro-enterostomy has been performed, the solar plexus has been stretched, and the dorsal spinal nerve roots of the seventh, eighth, ninth, and tenth have been divided with good results. The laryngeal crises are rarely dangerous. An application of cocaine may be made during the spasm, or a few whiffs of chloroform may be given, or nitrite of amyl. In all cases of tabes with increased arterial tension the prolonged use of nitroglycerin, given in increasing doses until the physiological effect is produced, is of great service in allaying the neuralgic pains and diminishing the frequency of the crises. Its use must be guarded when there is aortic insufficiency. The special indication is increased tension. The bladder symptoms demand constant care. When the organ can not be perfectly emptied the catheter should be used, and the patient may be taught its use and how to keep it thoroughly sterilized.

Frenkel's method of re-education often helps the patient to regain to a considerable extent the control of the voluntary movements which he has lost. By this method the patient is first taught, by repeated systematic efforts, to perform simple movements; from this he goes to more and more complex movements. The treatment should be directed and supervised by a trained teacher, as the result depends upon the skill of the teacher quite as much as upon the perseverance of the patient.

2. General Paralysis of the Insane and Tabo-paralysis

(*Dementia Paralytica; General Paresis*)

As has been said in the last section, the belief in the essential identity of general paralysis and tabes has gained more and more ground and has much in its favor. Mott says: "I maintain that etiologically and pathogenetically there is *one tabes* which may begin in the brain (especially in certain regions), or in the spinal cord in certain regions, or in the peripheral nervous structures connected with vision, or in nervous structures connected with the viscera, constituting, therefore, different types, any of which may be present or be associated with one or all of the others." Fournier has taken practically the same view and describes them together under the heading *Les Affections Parasyphilitiques.*

It is undoubted that most cases of tabes run their course with practically no mental symptoms, and that cases of general paralysis may never present symptoms that suggest tabes. For practical purposes we are forced to keep the distinction clearly in mind, and for this reason it seems best, at least for the present, to consider them separately.

There is, however, a group of cases in which the symptoms of the two diseases are associated in every combination. The name "tabo-paralysis" has been given to these cases.

General Paralysis

Definition.—A chronic, progressive disease of the brain and its meninges, associated with psychical and motor disturbances, finally leading to dementia and paralysis.

Etiology.—As in tabes, the important factor is syphilis, which is antecedent in both conditions in practically all cases. Males are affected much more frequently than females. It occurs chiefly between the ages of thirty and fifty-five, although it may begin in childhood as the result of congenital syphilis. An overwhelming majority of the cases are in married people, and not infrequently both husband and wife are affected, or one has paresis and the other tabes. Statistics show that it is more common in the lower classes of society, but in America in general medical practice the disease is certainly more common in the well-to-do classes. Heredity is a more important factor here than in tabes, although its influence is not great. An important predisposing cause is "a life absorbed in ambitious projects with all its strongest mental efforts, its long-sustained anxieties, deferred hopes, and straining expectation" (Mickle). The habits of life so frequently seen in active business men in our large cities, and well expressed by the phrase "burning the candle at both ends," strongly predispose to the disease.

Morbid Anatomy.—The dura is often thickened, and its inner surface may show the various forms of hypertrophic pachymeningitis. The pia is cloudy, thickened, and adherent to the cortex. The cerebro-spinal fluid is increased in the meningeal spaces, especially in the meshes of the pia, and at times to such an extent as to resemble cysts. The brain is small, and weighs less than normal. The convolutions are atrophied, especially in the anterior and middle lobes. In acute cases the brain may be swollen, hyperæmic, and œdematous. The brain cortex is usually red, and, except in advanced cases, it may not be atrophied, the atrophy of the hemispheres being at the expense of the white matter. The lateral ventricles are dilated to compensate for the atrophy of the brain, and the ependyma may be granular. The fourth ventricle is more constantly dilated, with granulations of its floor covering the calamus scriptorius, a condition seldom seen in any other affection.

The disease process is diffuse, and affects practically all parts of the brain, but its intensity varies greatly, even in adjoining areas. As a rule, the cortex of the frontal and central convolutions and the gray matter about the ventricles are most affected.

In many cases changes are present in the spinal cord and peripheral nerves. There are the typical tabetic changes described in the preceding section. There may be degeneration of the pyramidal systems of fibres secondary to the cortical changes. Most commonly there is a combination of these two processes. Foci of hæmorrhages, and softening dependent upon coarse vascular changes, are not infrequently found, but are not typical of the disease.

There are various views as to the nature of the changes. The vascular theory is that from an inflammatory process starting in the sheaths of the arterioles there is a diffuse parenchymatous degeneration with atrophic changes

Period cerebellar changes 12 years.
Mental 11th + 5th decades.

Progressive atrophy of centrum, especially of frontal lobes
fibrous leptomeningitis.

spinal cord normal in gross lesions - also nerves in spite of

Paralysis

dislocation of the shoulder

hemicrania. - [illegible] migraine. aneurism.

Temporary paresis. - [illegible] paral. + dislocation - [illegible]

-

Aphasia. Disturbance. mixes syllables or letters.

-

[illegible] any disturbance in region of [illegible]

tremor of tongue + hands - handwriting may be [illegible]

-

paralytic seizures. diagnosis - cerebral hemorrhage. (middle [illegible]

cerebral [illegible] - [illegible] - [illegible]

in the nerve cells and neuroglia. The most generally accepted view is that the syphilitic toxin causes degeneration in the nervous tissues with secondary changes in the neuroglia and vascular systems.

Symptoms.—Prodromal Stage.—This is of variable duration, and is characterized by a general mental state which finds expression in symptoms trivial in themselves but important in connection with others. Irritability, inattention to business amounting sometimes to indifference or apathy, and sometimes a *change in character*, marked by acts which may astonish the friends and relatives, may be the first indications. There may be unaccountable fatigue after moderate physical or mental exertion. Instead of apathy or indifference there may be an extraordinary degree of physical and mental restlessness. The patient is continually planning and scheming, or may launch into extravagances and speculation of the wildest character. A common feature at this period is the display of an unbounded egoism. He boasts of his personal attainments, his property, his position in life, or of his wife and children. Following these features are important indications of moral perversion, manifested in offences against decency or the law, many of which acts have about them a suspicious effrontery. Forgetfulness is common, and may be shown in inattention to business details and in the minor courtesies of life. At this period there may be no motor phenomena. The onset of the disease is usually insidious, although cases are reported in which epileptiform or apoplectiform seizures were the first symptoms. Among the early motor features are tremor of the tongue and lips in speaking, slowness of speech and hesitancy. Inequality of the pupils, the Argyll-Robertson pupil, optic atrophy, and changes in the deep reflexes may precede the occurrence of mental symptoms for years.

Second Stage.—This is characterized in brief by mental exaltation or excitement and a progress in the motor symptoms. "The intensity of the excitement is often extreme, acute maniacal states are frequent; incessant restlessness, obstinate sleeplessness, noisy, boisterous excitement, and blind, uncalculating violence especially characterize such states" (Lewis). It is at this stage that the delusion of grandeur becomes marked and the patient believes himself to be possessed of countless millions or to have reached the most exalted sphere possible in profession or occupation. This expansive delirium, as it is called, is, however, not characteristic, as was formerly supposed, of paralytic dementia. Besides, it does not always occur, but in its stead there may be marked melancholia or hypochondriasis, or, in other instances, alternate attacks of delirium and depression.

The facies has a peculiar stolidity, and in speaking there is marked tremulousness of the lips and facial muscles. The tongue is also tremulous, and may be protruded with difficulty. The speech is slow, interrupted, and blurred. Writing becomes difficult on account of unsteadiness of the hand. Letters, syllables, and words may be omitted. The subject matter of the patient's letters gives valuable indications of the mental condition. In many instances the pupils are unequal, irregular, sluggish, sometimes large. Important symptoms in this stage are apoplectiform seizures and paralysis. There may be slight syncopal attacks in which the patient turns pale and may fall. Some of these are *petit mal*. In the true apoplectiform seizure the patient falls suddenly, becomes unconscious, the limbs are relaxed, the face is flushed, the breathing stertorous, the temperature increased, and death may occur. Epileptic seizures

are more common than the apoplectiform. There may be a definite aura. The attack usually begins on one side and may not spread. There may be twitchings either in the facial or brachial muscles. Typical Jacksonian epilepsy may occur. Recurring attacks of aphasia are not uncommon, and paralysis, either monoplegic or hemiplegic, may follow these epileptic seizures, or may come on with great suddenness and be transient. In this stage the gait becomes impaired, the patient trips readily, has difficulty in going up or down stairs, and the walk may be spastic or occasionally tabetic. This paresis may be progressive. The deep reflexes are usually increased, but may be lost. Bladder or rectal symptoms gradually develop. The patient becomes helpless, bedridden, and completely demented, and unless care is taken may suffer from bedsores. Death occurs from exhaustion or from some intercurrent affection. The spinal cord features of dementia paralytica may come on with or precede the mental troubles. There are cases in which one is in doubt for a time whether the symptoms indicate tabes or dementia paralytica, and it is well to bear in mind that every feature of pre-ataxic tabes may exist in the early stage of general paresis.

Tabo-paralysis

Emphasis has been laid on the ~~probable~~ identity of the processes underlying tabes and ~~dementia paralytica~~, the spinal cord in the first case receiving the full force of the attack, and the brain in the second. ~~It has been thought that~~ stress ~~is the factor which~~ determines the location of the process, ~~and that~~ men whose occupations require much bodily exercise ~~would be~~ apt to have tabes, ~~while~~ those whose activities are largely mental ~~would~~ suffer from paresis. Usually when the cord symptoms are pronounced the symptoms from the brain remain in abeyance, and the reverse is also true. There are exceptions ~~to this~~, and cases of well marked tabes may later show the typical symptoms of paresis, but even then the ataxia, if it is not of too high a grade, ~~often~~ improves.

Optic atrophy, when it occurs in the pre-ataxic stage of tabes, usually indicates that the ataxia will never be pronounced, but unfortunately it is frequently followed by the occurrence of mental symptoms. Mott ~~believes~~ that about 50 per cent. of his asylum cases of tabo-paralysis had had preceding optic atrophy. Its occurrence is therefore of grave significance. The mental symptoms may be delayed for many years.

The *symptom complex* of tabo-paralysis ~~is~~ made up of a combination of ~~the symptoms~~ of the two conditions ~~and~~ varies greatly. It may begin as tabes with lightning pains, bladder symptoms, Argyll-Robertson pupil, loss of the deep reflexes, etc., to have the mental symptoms added later; or, on the other hand, cord symptoms may come on after the patient has shown marked mental changes. ~~In a number of cases~~ the symptoms ~~are~~ from the first so combined that the name tabo-paralysis is at once applicable. Absent knee-jerks, ocular palsies, or pupillary symptoms may precede the breakdown for many years, but none of them have so grave a significance in regard to the mental state as has optic atrophy. Other types of alienation may interrupt the course of tabes, and the mistake must not be made of regarding them all as general paralysis. In such instances the mind may become clear and remain so to the end.

Diagnosis.—The recognition of general paralysis in the earliest stage is

paralytic psychosis

depressive-
expansive-
agitated.
demented.

colloidal gold reaction in Spinalfluid. 48-100%

Lymphocytose in Spinalfluid. 48%

[illegible]

Blutsenkung. 48-100%

Sp.-fluid " 57%

positive Globulin. 100%

extremely difficult, as it is often impossible to decide that the slight alteration in conduct is anything more than one of the moods or phases to which most men are at times subject. The following description by Folsom is an admirable presentation of the diagnostic characters of the early stage of the disease: "It should arouse suspicion if, for instance, a strong, healthy man, in or near the prime of life, distinctly not of the 'nervous,' neurotic, or neurasthenic type, shows some loss of interest in his affairs or impaired faculty of attending to them; if he becomes varyingly absent-minded, heedless, indifferent, negligent, apathetic, inconsiderate, and, although able to follow his routine duties, his ability to take up new work is, no matter how little, diminished; if he can less well command mental attention and concentration, conception, perception, reflection, judgment; if there is an unwonted lack of initiative, and if exertion causes unwonted mental and physical fatigue; if the emotions are intensified and easily change, or are excited readily from trifling causes; if the sexual instinct is not reasonably controlled; if the finer feelings are even slightly blunted; if the person in question regards with a placid apathy his own acts of indifference and irritability and their consequences, and especially if at times he sees himself in his true light and suddenly fails again to do so; if any symptoms of cerebral vaso-motor disturbances are noticed, however vague or variable."

There are cases of cerebral syphilis which closely simulate dementia paralytica. The mode of onset is important, particularly since paralytic symptoms are usually early in syphilis. The affection of the speech and tongue is not present. Epileptic seizures are more common and more liable to be cortical or Jacksonian in character. The expansive delirium is rare. While symptoms of general paresis are not common in connection with the development of gummata or definite gummatous meningitis, there are, on the other hand, instances of paresis following closely upon the syphilitic infection. Post mortem in such cases there may be nothing more than a general arteriosclerosis and diffuse meningo-encephalitis, which may present nothing distinctive, but the lesions, nevertheless, may be caused by the syphilitic virus. Cases also occur in which typical syphilitic lesions are combined with the ordinary lesions of dementia paralytica. There are certain forms of lead encephalopathy which resemble general paresis, and, considering the association of plumbism with arterio-sclerosis, it is not unlikely that the anatomical substratum of the disease may result from this poison. Tumor may sometimes simulate progressive paresis, but in the former the signs of general increase of the intracranial pressure are usually present. The Wassermann reaction in the blood or spinal fluid is nearly constant.

Cytodiagnosis.—The study of the cellular elements ~~suspended~~ in the cerebro-spinal fluid ~~has come to be~~ an important diagnostic measure, particularly in tabes and paresis. In both of these affections spinal lymphocytosis is the rule and is usually associated with a marked albumin reaction—the normal fluid containing no albumin, or at most minute traces, and a negligible number of formed elements. It is simply the expression of a subacute or chronic inflammatory process, just as polymorphonuclear leukocytosis is characteristic of an acute process. It is, however, first and foremost the syphilitic triad—tabes, paresis, and cerebro-spinal lues—which is suggested by lymphocytosis in the spinal fluid. Positive reactions, cytological and chemical, are

among the earliest somatic symptoms, and may therefore clear up obscure cases of tabes and paresis, just at the time when diagnosis is most difficult.

Prognosis.—The disease rarely ends in recovery. As a rule the progress is slowly downward and the case terminates in a few years, although it is occasionally prolonged ten or fifteen years.

Treatment.—Salvarsan in small repeated doses may be tried in early cases, after which large doses of iodide of potassium and a mercurial course should be given. Careful nursing and the orderly life of an asylum are the only measures necessary in a great majority of the cases. For sleeplessness and the epileptic seizures bromides may be used. Prolonged remissions, which are not uncommon, are often erroneously attributed to the action of remedies. Active treatment in the early stage by wet-packs, cold to the head, and systematic massage has been followed by temporary improvement.

3. ACUTE POSTERIOR GANGLIONITIS

(*Herpes Zoster*)

Definition.—An acute disease with localization in the cerebral ganglia and in the ganglia of the posterior nerve roots, associated with a vesicular inflammation of the skin of the corresponding cutaneous areas.

Distribution.—Herpes most frequently occurs in the region of the dorsal roots and extends in the form of a half girdle, on which account the names "zona" and "zoster" have been given. The trigeminal region is very often involved, particularly the first branch. Common forms also are the herpes sterno-nuchalis, cervico-subclavicularis and dorso-ulnaris.

Pathology.—Bärensprung first showed that there was involvement of the spinal ganglia. The exhaustive studies of Head and Campbell show that the primary disease is an acute hæmorrhagic inflammation of the ganglia of the posterior nerve roots and of the homologous cranial ganglia. It is analogous to acute anterior polio-myelitis. There are inflammatory foci, hæmorrhage in and destruction of certain of the ganglion cells leading to degeneration of the axis-cylinders. In herpes facialis accompanying pneumonia W. T. Howard has shown that similar lesions are demonstrable in the Gasserian ganglion, and Hunt has found the same changes in the otic ganglion in herpes auricularis. It is met with in the acute infections, particularly pneumonia, malaria, and cerebro-spinal fever. Epidemics have been described. Micro-organisms have been found in the cerebro-spinal fluid.

Symptoms.—In ordinary zona there is often a slight prodromal period in which the patient feels ill, has moderate fever, and pain in the side, sometimes of such severity as to suggest pleurisy. On the third or fourth day the rash appears. The characteristic group of vesicles has a segmental distribution limited to one side of the body. One or more of the adjoining skin fields is usually affected. With involvement of the cervical, lumbar, or sacral ganglion the zonal or girdle form of the vesicular crop is naturally lost owing to the distortion of the skin fields from the growth of the limbs. The typical zonal form is only seen in involvement of the thoracic ganglia. Groups of vesicles are regularly arranged on the hyperæmic skin, at first filled with a clear or sometimes bloody serum, which later becomes purulent. The crop varies greatly, and the individual vesicles may be superficial, in which case

Rosenow – ganglionites in acromias with herpes –

sinusite do ganglion (fracture) or tumors

they leave no scar, or they may be deep and in healing leave superficial scars. By far the most serious form is that seen in the upper division of the fifth. The fever may be high and the eruption very profuse with great swelling and much pain. ~~I have seen several cases of~~ permanent disfigurement ~~from~~ the scarring.

It seems not improbable, as Chauffard suggests, that there may be extension of the disease from the posterior ganglia to the neighboring meninges as there may be pains down the spine, the girdle sensation, exaggerated knee-jerks, the Kernig sign, and lymphocytosis in the cerebro-spinal fluid.

Complications.—Perhaps the most serious ~~of these~~ is that occasionally seen in ophthalmic zoster, when there is intense inflammation of the conjunctiva and cornea with consecutive pan-ophthalmitis and destruction of the eye, ~~of which I have seen one instance.~~

In a few cases the eruption becomes gangrenous. Swelling of the lymph glands has been noted. A bilateral distribution has occurred. A generalized herpes zoster is occasionally seen with a widespread vesicular rash on the face, neck, trunk, and thighs. A facial paralysis may develop during or after ophthalmic or cervical herpes. ~~I have seen~~ swelling of the parotid gland on the same side. In rare cases paralysis of the extremities has occurred. By far the most distressing complication is *post-zonal neuralgia.* After recovery from the herpes, hot burning sensations are not uncommon in the cutaneous distribution. In other instances, particularly in old people, the pain persists and for years may be a terrible affliction resisting all ~~measures of~~ treatment. ~~Gowers speaks of~~ suicide in despair, ~~one instance of which came under my notice.~~

Treatment.—Care should be taken to protect the vesicles; a one per cent. cocaine ointment with lanolin carefully applied on lint gives relief to the pain. In very severe involvement of the ophthalmic division of the fifth nerve the greatest care should be taken to keep the conjunctiva clean. For the severe post-zonal neuralgia, injections into the spinal cord have been tried, and in cases of great severity the posterior nerve roots may be cut.

III. DISEASES OF THE EFFERENT OR MOTOR TRACT

A. OF WHOLE TRACT

1. PROGRESSIVE (CENTRAL) MUSCULAR ATROPHY

(Poliomyelitis Anterior Chronica; Amyotrophic Lateral Sclerosis; Progressive Bulbar Paralysis)

Definition.—A disease characterized by a chronic degeneration of the motor tract, usually of the whole, but at times limited to the lower segment. Associated with it is a progressive atrophy of the muscles, with more or less spastic rigidity.

Three affections, as a rule described apart, belong together in this category: (*a*) Progressive muscular atrophy of spinal origin; (*b*) amyotrophic lateral sclerosis; and (*c*) progressive bulbar paralysis. A slow atrophic change in the motor neurones is the anatomical basis, and the disease is one of the whole

motor path, involving, in many cases, the cortical, bulbar, and spinal centres. There may be simple muscular atrophy with little or no spasm, or progressive wasting with marked spasm and great increase in the reflexes. In others, there are added symptoms of involvement of the motor nuclei in the medulla—a glosso-labio-laryngeal paralysis; while in others, again, with atrophy (especially of the arms), a spastic condition of the legs and bulbar phenomena, tremors develop and signs of cortical lesion. These various stages may be traced in the same case.

For convenience, bulbar paralysis will be considered separately, and here are taken together *progressive muscular atrophy* and *amyotrophic lateral sclerosis.*

The disease is known as the Aran-Duchenne type of progressive muscular atrophy and as Cruveilhier's palsy, after the French physician who early described it. Luys and Lockhart Clarke first demonstrated that the cells of the ventral horns of the spinal cord were diseased. Charcot separated two types—one with simple wasting of the muscles, due, he believed, to degeneration confined to the ventral horns (and to this he restricted the name progressive muscular atrophy—type, Aran-Duchenne); the other, in which there was spastic paralysis of the muscles followed by atrophy. As the anatomical basis for this he assumed a primary degeneration of the pyramidal tracts and a secondary atrophy of the ventral horns. To this he gave the name of amyotrophic lateral sclerosis. There is but little evidence, however, to show that any such sharp distinction can be made between these two diseases, and Leyden and Gowers regard them as identical.

Etiology.—The cause ~~of the disease~~ is unknown. It is more frequent in males than in females. It affects adults, usually after the thirtieth year, though occasionally younger persons are attacked. ~~A majority of all~~ cases of progressive muscular atrophy under twenty five years of age belong to the dystrophies. Cold, wet, exposure, fright, and mental worries are mentioned as possible causes. Certain cases follow injury. ~~Hereditary influences are present in some cases.~~ The rare form which occurs in infancy ~~usually~~ affects ~~several members of the same family.~~ Hereditary and family influences, however, play but a small part in the etiology, and in this it is in contrast to progressive neural muscular atrophy and the dystrophies. Yet, in the Farr family, which I recorded ~~some years ago~~, in which thirteen members were affected in two generations, with the exception of two, the cases occurred or proved fatal above the age of forty, and the late onset speaks rather for a central affection. The spastic form may develop late in life—after seventy—as a senile change.

Morbid Anatomy.—The essential anatomical change is a slow degeneration of the motor path, involving particularly the lower motor neurones. The upper neurones are also involved, either first, simultaneously, or at a later period. Associated with the degeneration in the cells of the ventral horns there is a degenerative atrophy of the muscles. The following are the important anatomical changes: (*a*) The gray matter of the cord shows the most marked alteration. The large ganglion cells of the ventral horns are atrophied, or, in places, have entirely disappeared, the neuroglia is increased, and the medullated fibres are much decreased. The fibres of the ventral nerve-roots passing through the white matter are wasted. (*b*) The ventral roots outside of the

Wernicke-Hoffman type is a familial affection and does not belong here.

parenchym. degeneration of the nervous with fibrillation of the muscular tissues.

cord are also atrophied. (*c*) The muscles affected show degenerative atrophy, and the inter-muscular branches of the motor nerves are degenerated. (*d*) The degeneration of the gray matter is rarely confined to the cord, but extends to the medulla, where the nuclei of the motor cerebral nerves are found extensively wasted. (*e*) In a majority of all the cases there is sclerosis in the ventro-lateral white tracts, the lateral pyramidal tracts particularly are diseased, but the degeneration is not confined to them, but extends into the ventro-lateral ground bundles. The direct cerebellar and the ventro-lateral ascending tracts are spared. The degeneration in the pyramidal tracts extends toward the brain to different levels, and in several cases has been traced to the motor cortex, the cells of which have been found degenerated. In the medulla the medial longitudinal fasciculus has been found diseased. (*f*) In those cases in which no sclerosis has been found in the pyramidal tracts there has been a **sclerosis of the ventro-lateral ground bundle (short tracts).**

Symptoms.—Irregular pains may precede the onset of the wasting, and cases may be treated for "chronic rheumatism." The hands are usually first affected, and there is difficulty in performing delicate manipulations. The muscles of the ball of the thumb waste early, then the interossei and lumbricales, leaving marked depressions between the metacarpal bones. Ultimately the contraction of the flexor and extensor muscles and the extreme atrophy of the thumb muscles, the interossei, and lumbricales produce the claw-hand —*main en griffe* of Duchenne. The flexors of the forearm are usually involved before the extensors. In the shoulder-girdle the deltoid is first affected; it may waste even before the other muscles of the upper extremity. The trunk muscles are gradually attacked; the upper part of the trapezius long remains unaffected. Owing to the feebleness of the muscles which support it, the head tends to fall forward. The platysma myoides is unaffected and often hypertrophies. The arms and the trunk muscles may be much atrophied before the legs are attacked. The face muscles are attacked late. Ultimately the intercostal and abdominal muscles may be involved, the wasting proceeds to an extreme grade, and the patient may be actually "skin and bone," and, as "living skeletons," the cases are not uncommon in "museums" and "side-shows." Deformities and contractures result, and lordosis is almost always present. A curious twitching of the muscles (fibrillation) is a common symptom, and may occur in muscles which are not yet attacked. It is a most important symptom, but is not, as was formerly supposed, a characteristic feature. The irritability of the muscles is increased. Sensation is unimpaired, but the patient may complain of numbness and coldness of the affected limbs. The galvanic and faradic irritability of the muscles progressively diminishes and may become extinct, the galvanic persisting for the longer time. In cases of rapid wasting and paralysis the reaction of degeneration may be obtained. The excitability of the nerve trunks may persist after the muscles have ceased to respond. The loss of power is usually proportionate to the wasting.

The foregoing description applies to the group of cases in which the atrophy and paralysis are flaccid—*atonic*, as Gowers calls it. In other cases, those which Charcot describes as amyotrophic lateral sclerosis, spastic paralysis precedes the wasting. This *tonic* atrophy first involves the arms and then the legs. The reflexes are greatly increased. It is one of the rare conditions in which a jaw clonus may be obtained. The most typical condition of spastic

paraplegia may be produced. On starting to walk, the patient seems glued to the ground and makes ineffectual attempts to lift the toes; then four or five short, quick steps are taken on the toes with the body thrown forward; and finally he starts off, sometimes with great rapidity. Some of the patients can walk up and down stairs better than on the level. The wasting is never so extreme as in the atonic form, and the loss of power may be out of proportion to it. The sphincters are unaffected. Sexual power may be lost early. Cases are met with which correspond accurately to the clinical picture given by Charcot of amyotrophic lateral sclerosis. These are not very common, and it is much more usual to have a combination of the two types. A flaccid atrophic paralysis with increased reflexes is often met with. These differences depend upon the relative extent of the involvement of the upper and lower motor segments and the time of the involvement of each. The condition may be unilateral.

As the degeneration extends upward an important change takes place from the occurrence of bulbar symptoms, which may, however, precede the spinal manifestations. The lips, tongue, face, pharynx, and larynx may be involved. The lips may be affected and articulation impaired for years before serious symptoms occur. In the final stage there may be tremor, the memory fails, and a condition of dementia supervenes.

Gowers gives the following useful classification of the varieties of this affection: (1) Atonic atrophy, becoming extreme; (2) muscular weakness with spasm, but without wasting or with only slight wasting; and (3) atonic atrophy, rarely extreme in degree, with exaggeration of the reflexes. These conditions may "coexist in every degree and combination—between universal atonic atrophy on the one hand and universal spastic paralysis without wasting on the other."

Diagnosis.—Progressive (central) muscular atrophy begins, as a rule, in adult life, without hereditary or family influences (the early infantile form being an exception), and usually affects first the muscles of the thumb, and gradually involves the interossei and lumbricales. Fibrillary contractions are common, electrical changes occur, and the deep reflexes are usually increased. These characteristics are usually sufficient to distinguish it from the other forms of muscular wasting.

In syringomyelia the symptoms may be very similar to those in the spastic form of muscular atrophy. The sensory disturbances in the former disease, as a rule, make the diagnosis clear, but when these are absent or but little developed it may be very difficult or even impossible to distinguish the diseases.

Treatment.—The disease is incurable. The downward progress is slow but certain, though in a few cases a temporary arrest may take place. With a history of syphilis, mercury and iodide of potassium may be tried, and Gowers recommends courses of arsenic and the hypodermic injection of strychnine. Probably the most useful means is systematic massage, particularly in the spastic cases.

Bulbar Paralysis (*Glosso-labio-laryngeal Paralysis*)

When the disease affects the motor nuclei of the medulla first or early, it is called bulbar paralysis, but it has practically no independent existence, as the spinal cord is sooner or later involved.

Symptoms.—The disease usually begins with slight defect in the speech, and the patient has difficulty in pronouncing the dentals and linguals. The paralysis starts in the tongue, and the superior lingual muscle gradually becomes atrophied, and finally the mucous membrane is thrown into transverse folds. In the process of wasting the fibrillary tremors are seen. Owing to the loss of power in the tongue, the food is with difficulty pushed back into the pharynx. The saliva also may be increased, and is apt to accumulate in the mouth. When the lips become involved the patient can neither whistle nor pronounce the labial consonants. The mouth looks large, the lips are prominent, and there is constant drooling. The food is masticated with difficulty. Swallowing becomes difficult, owing partly to the regurgitation into the nostrils, partly to the involvement of the pharyngeal muscles. The muscles of the vocal cords waste and the voice becomes feeble, but the laryngeal paralysis is rarely so extreme as that of the lips and tongue.

The **course** of the disease is slow but progressive. Death often results from an aspiration pneumonia, sometimes from choking, more rarely from involvement of the respiratory centres. The mind usually remains clear. The patient may become emotional. In a majority of the cases the disease is only part of a progressive atrophy, either simple or associated with a spastic condition. In the later stage of amyotrophic lateral sclerosis the bulbar lesions may paralyze the lips long before the pharynx or larynx becomes affected.

The **diagnosis** of the disease is readily made, either in the acute or chronic form. The involvement of the lips and tongue is usually well marked, while that of the palate may be long deferred. A condition has been described, however, which may closely simulate bulbar paralysis. This is the so-called *pseudo-bulbar* form or bulbar palsy of cerebral origin. Bilateral disease of the motor cortex in the lower part of the ascending frontal convolution, or about the knee of the internal capsule, may cause paralysis of the lips and tongue and pharynx, which closely simulates a lesion of the medulla. Sometimes the symptoms appear on one side, but in many instances they develop suddenly on both sides. A bilateral lesion has usually been found, but in several instances the disease was unilateral.

The so-called *acute bulbar paralysis* may be due to (*a*) hæmorrhagic or embolic softening in the pons and medulla; (*b*) acute inflammatory softening, analogous to polio-myelitis, occurring occasionally as a post-febrile affection. It has occasionally followed diphtheria, and occurred after severe electric shocks of high voltage. It usually comes on very suddenly, hence the term apoplectiform. The symptoms in this form may correspond closely to those of an advanced case of chronic bulbar paralysis. The sudden onset and the associated symptoms make the diagnosis easy. In these acute cases there may be loss of power in one arm, or hemiplegia, sometimes alternate hemiplegia, with paralysis on one side of the face and loss of power on the other side of the body. (*c*) In epidemics of polio-myelitis cases occur with acute bulbar symptoms.

2. PROGRESSIVE NEURAL MUSCULAR ATROPHY

This form, known also as the peroneal type, or by the names of the men who have described it most accurately—namely, Charcot, Marie, and Tooth—

occurs either as a hereditary or as a family affection. It usually begins in early childhood, affecting first the muscles of the feet and the peroneal group; as a result of the weakening of these muscles, club-foot, either pes equinus or pes equino-varus, occurs. In rare instances the disease may begin in the hands, but the upper limbs, as a rule, are not affected for some years after the legs are attacked, and the trouble then begins in the small muscles of the hand. Sensory disturbances are frequently present and form important diagnostic features. Fibrillary contractions and twitchings also occur. The electrical reactions are altered; there is either a loss or a very great decrease of the excitability, which can be demonstrated not only in the atrophic muscles, but also in muscles and nerves which are apparently normal.

This form seems to stand between the central muscular atrophy and the muscular dystrophies. Occurring in families and beginning in early life, it resembles the latter, but it is more like the former in that fibrillary contractions and muscular twitchings are common, that the small muscles of the hand are apt to be involved, and that electrical changes are present. In the prominence of sensory symptoms it differs from both. In cases of acquired double club-foot this disease should be suspected.

3. THE MUSCULAR DYSTROPHIES

(*Dystrophia muscularis progressiva,* Erb)

Definition.—Muscular wasting, with or without an initial hypertrophy, beginning in various groups of muscles, usually progressive in character, and dependent on primary changes in the muscles themselves. A marked hereditary disposition is met with in the disease.

Etiology.—No etiological factors of any moment are known other than heredity. The influence may show itself by true heredity—the disease occurring in two or more generations—or several members of the same generation may be affected, showing a family tendency. Many members of the same family may be attacked through several generations. Males, as a rule, are more frequently affected than females. The disease is usually transmitted through the mother, though she may not herself be affected. As many as 20 or 30 cases have been described in five generations. In Erb's cases 44 per cent. showed no heredity. The disease usually sets in before puberty, but may be as late as the twentieth or twenty-fifth year, or in some instances even later.

Symptoms.—The first symptom noticed is, as a rule, clumsiness in the movements of the child, and on examination certain muscles or groups of muscles seem to be enlarged, particularly those of the calves. The extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps and infraspinatus, are the next most frequently involved, and may stand out with great prominence. The muscles of the neck, face, and forearm rarely suffer. Sometimes only a portion of a muscle is involved. With this hypertrophy of some muscles there is wasting of others, particularly the lower portion of the pectorals and the latissimus dorsi. The attitude when standing is very characteristic. The legs are far apart, the shoulders thrown back, the spine is greatly curved, and the abdomen protrudes. The gait is waddling and awkward. In

Tendon reflexes decrease and are absent finally. The skin reflexes are usually normal

getting up from the floor the position assumed, so well known now through Gowers' figures, is pathognomonic. The patient first turns over in the all-fours position and raises the trunk with his arms; the hands are then moved along the ground until the knees are reached; then with one hand upon a knee he lifts himself up, grasps the other knee, and gradually pushes himself into the erect posture, as it has been expressed, by climbing up his legs. The striking contrast between the feebleness of the child and the powerful looking pseudo-hypertrophic muscles is very characteristic. The enlarged muscles may, however, be relatively very strong.

The course of the disease is slow, but progressive. Wasting proceeds and finally all traces of the enlarged condition of the muscles disappear. At this late period distortions and contractions are common.

The muscles of the shoulder-girdle are nearly always affected early in the disease, causing a symptom upon which Erb lays great stress. With the hands under the arms, when one endeavors to lift the patient, the shoulders are raised to the level of the ears, and one gets the impression as though the child were slipping through. These "loose shoulders" are very characteristic. The abnormal mobility of the shoulder blades gives them a winged appearance, and makes the arms seem much longer than usual when they are stretched out.

There are no sensory symptoms. The atrophic muscles do not show the reaction of degeneration except in extremely rare instances.

Clinical Forms.—A number of different types have been described, depending upon the age at the onset, the muscles first affected, the occurrence of hypertrophy, the prominence of heredity, etc. But Erb has shown that there is no sharp division between these different forms, and classes them all under the name of *dystrophia muscularis progressiva*. For convenience of description he subdivides the disease into two large groups:

I. Those cases which occur in childhood.

II. The cases occurring in youth and adult life.

The first division is subdivided into (1) the hypertrophic and (2) the atrophic form.

Under the hypertrophic form, which is the pseudo-hypertrophic muscular paralysis of authors, he thinks it is useful to distinguish between the cases in which (*a*) the enlarged muscles have undergone lipomatosis—i. e., pseudo-hypertrophy—from those (*b*) in which there is a real hypertrophy.

The atrophic form also includes two subclasses: (*a*) Those cases in which the muscles of the face are involved early; this corresponds to the infantile form of Duchenne—the Landouzy-Dejerine type. (*b*) Those cases in which the face is not involved. They may be grouped as follows:

I. *Dystrophia muscularis progressiva infantum.*

1. Hypertrophic form.
   - (*a*) With pseudo-hypertrophy.
   - (*b*) With real hypertrophy.
2. Atrophic form.
   - (*a*) With primary involvement of the face (infantile form of Duchenne).
   - (*b*) Without involvement of the face.

II. *Dystrophia muscularis progressiva juvenum vel adultorum* (Erb's juvenile form).

Morbid Anatomy.—According to Erb, the disease consists in a change in the muscles themselves. At first the muscle fibres hypertrophy, and become round; the nuclei increase, and the muscle fibres may become fissured. At the same time there is a slight increase in the connective tissue. Sooner or later the muscle fibres begin to atrophy, and the nuclei become greatly increased. Vacuoles and fissures appear, and the fibres finally become completely atrophic, the connective tissue becoming markedly increased. Fat may be deposited in the connective tissue to such an extent as to cause hypertrophic lipomatosis—pseudo-hypertrophy. The different stages of these changes may be found in a single muscle at the same time.

The nervous system has very generally been found to be without demonstrable lesions, but in certain cases changes in the cells of the ventral horns have been described.

Diagnosis.—The muscular dystrophies can usually be distinguished readily from the other forms of muscular atrophy.

(*a*) In the cerebral atrophy loss of power usually precedes the atrophy, which is either of a monoplegic or hemiplegic type.

(*b*) From progressive (central) muscular atrophy the distinctions are plainly marked. This form begins in the small muscles of the hand, a situation rarely, if ever, affected by the dystrophies, which involve first those of the calves, the trunk, the face, or the shoulder-girdle. In the central atrophy the reaction of degeneration is present and fibrillary twitchings occur in both the atrophied and non-atrophied muscles. In many cases, in addition to the wasting in the arms, there is a spastic condition in the legs and increase in the reflexes. The central atrophies come on late in life; the dystrophies develop, as a rule, early. In the progressive muscular dystrophies heredity plays an important rôle, which in the central form is quite subsidiary. In the rare cases of early infantile spinal muscular atrophy occurring in families the symptoms are so characteristic of a central disease that the diagnosis presents no difficulty.

(*c*) In the neuritic muscular atrophies, whether due to lead or to trauma, the general characters and the mode of onset are distinctive. In the cases of multiple neuritis seen for the first time at a period when the wasting is marked there is often difficulty, but the absence of family history and the distribution are important features. Moreover, the paralysis is out of proportion to the atrophy. Sensory symptoms may be present, and in the cases in which the legs are chiefly involved there is usually the *steppage* gait so characteristic of peripheral neuritis.

(*d*) Progressive neural muscular atrophy. Here heredity is also a factor, and the disease usually begins in early life, but the distribution of atrophy and paralysis, which in this affection is at first confined to the periphery of the extremities, helps to distinguish it from the dystrophies; while the occurrence of sensory symptoms, fibrillary contractions, and the marked decrease in the electrical excitability usually make the distinction clear.

The outlook in the primary muscular dystrophies is bad. The wasting progresses uniformly, uninfluenced by treatment. Erb holds that by electricity and massage the progress is occasionally arrested. The general health should be carefully looked after, moderate exercise allowed, frictions with oil applied to the muscles, and when the patient becomes bedfast, as is inevitable

sooner or later, care should be taken to prevent contractures in awkward positions.

The forms of progressive muscular wasting—progressive (central) muscular atrophy, progressive neural muscular atrophy, and the muscular dystrophies—have been considered as distinct diseases, but possibly the distinction may not be so sharp as we believe. Certain cases occur which seem not to belong to any one of the forms, but to stand between them. The changes in the muscles which were thought to be characteristic of the dystrophies have been found in the other forms. The central form occurs as a family disease in infancy, and the nervous system has been found diseased in the dystrophies.

B. SYSTEM DISEASES OF THE UPPER MOTOR SEGMENT

The question of an uncomplicated primary degeneration of the upper motor neurones has not been decided. Cases with a clinical picture corresponding to this lesion are not uncommon, and they may persist for a long time without change. Unfortunately the cases which have come to autopsy have shown various conditions. In only two or three has the disease been so nearly confined to the pyramidal tract that they can be used as an argument for the independence of this condition. The cases of Minkowski, Dreschfeld, and Strümpell are not absolutely conclusive, as they are not quite pure, although they go far to prove that a degeneration in the pyramidal tract may be uncomplicated, at least for a long time. The same may be said for the group of cases described by Bernhardt and Strümpell under the name hereditary spastic spinal paralysis, in which the extensive systemic degeneration of the pyramidal tracts is combined with slight degeneration in other tracts of the cord.

1. SPASTIC PARALYSIS OF ADULTS

(*Tabes dorsalis spasmodique; Primary Lateral Sclerosis*)

Definition.—A gradual loss of power with spasm of the muscles of the body, the lower extremities being first and most affected, unaccompanied by muscular atrophy, sensory disturbance, or other symptoms. The pathological anatomy is undetermined, but a systemic degeneration of the pyramidal tracts is assumed.

Symptoms.—The general symptoms of spastic paraplegia in adults are very distinctive. The patient complains of feeling tired, of stiffness in the legs, and perhaps of pains of a dull aching character in the back or in the calves. There may be no definite loss of power, even when the spastic condition is well established. In other instances there is definite weakness. The stiffness is felt most in the morning. In a well developed case the gait is most characteristic. The legs are moved stiffly and with hesitation, the toes drag and catch against the ground, and, in extreme cases, when the ball of the foot rests upon the ground a distinct clonus develops. The legs are kept close together, the knees touch, and in certain cases the adductor spasm may cause cross-legged progression. On examination, the legs may at first appear

tolerably supple, perhaps flexed and extended readily. In other cases the rigidity is marked, particularly when the limbs are extended. The spasm of the adductors of the thigh may be so extreme that the legs are separated with the greatest difficulty. In cases of this extreme rigidity the patient usually loses the power of walking. The nutrition is well maintained, the muscles may be hypertrophied. The reflexes are greatly increased. The slightest touch upon the patellar tendon produces an active knee-jerk. The rectus clonus and the ankle clonus are easily obtained. In some instances the slightest touch may throw the legs into violent clonic spasm, the condition to which Brown-Séquard gave the name of spinal epilepsy. The superficial reflexes are also increased. The arms may be unaffected for years, but occasionally they become weak and stiff at the same time as the legs.

The course of the disease is progressively downward. Years may elapse before the patient is bedridden. Involvement of the sphincters, as a rule, is late; occasionally, however, it is early. The sensory symptoms rarely progress, and the patients may retain their general nutrition and enjoy excellent health. Ocular symptoms are rare.

Diagnosis.—The diagnosis, so far as the clinical picture is concerned, is readily made, but it is often very difficult to determine accurately the nature of the underlying pathological condition. A history of syphilis is present in many of the cases. Cases which have run a fairly typical clinical course upon coming to autopsy have been found to have been due to very different conditions—transverse myelitis, multiple sclerosis, cerebral tumor, etc. General paralysis of the insane may begin with symptoms of spastic paraplegia, and Westphal believed that it was only in relation to this disease that a primary sclerosis of the pyramidal tracts ever occurred. In any case the diagnosis of primary systemic degeneration of the pyramidal tract is, to say the least, doubtful.

Treatment.—Not much can be done to check the progress of the disease. Division of the posterior nerve roots is permissible when the motor weakness is due chiefly to the spasticity. A number of cases have been operated upon successfully. The same practice has been followed in the spasticity with bilateral athetosis.

2. SPASTIC PARALYSIS OF INFANTS—SPASTIC DIPLEGIA—BIRTH PALSIES

(*Paraplegia cerebralis spastica* (*Heine*); *Little's Disease*)

In this condition there is a paralysis with spasm of all extremities, dating from or shortly succeeding birth, more rarely following the fevers or an attack of convulsions. The legs are usually more involved than the arms; there is no wasting, no disturbance of sensation. The reflexes are increased. The mental condition is usually much disturbed. The patients are often imbeciles or idiots, helpless in mind and body. Ataxic and athetoid movements of the most exaggerated kind may occur.

While only a limited number of cases of infantile hemiplegia are congenital, on the other hand, in spastic diplegia and paraplegia a large proportion of the cases results from injury at birth. The arms may be so slightly affected as to make it difficult to determine whether it is a case of diplegia or

paraplegia. The disease usually dates from birth, and a majority of the children are born in first labors or are forceps cases, and are at birth asphyxiated blue babies. In feet presentations there may be laceration or tearing of the cerebro-spinal membranes. Premature birth is also given as a cause.

Morbid Anatomy.—The birth palsies which ultimately induce the spastic diplegias or paraplegias are most frequently the result of meningeal hæmorrhage. The importance of this condition has been shown by the studies of Litzmann and Sarah J. McNutt. The bleeding may come from the veins, or from the longitudinal sinus. The hæmorrhage has in many cases been greatest over the motor areas, and in these cases the intelligence may suffer but little; with a more extensive hæmorrhage, especially when it implicates the frontal lobes, any grade of amentia may be occasioned. It seems probable that the sclerosis found in these cases may result from compression by the blood clot. In other instances the condition may be due to a fetal meningo-encephalitis. In 16 autopsies collected in the literature, in which the patients died at ages varying from two to thirty, the anatomical condition was either a diffuse atrophy, which was most common, or porencephalus. From the fact that certain of the patients are born prematurely, before the pyramidal tracts are developed, it has been assumed by some that a non-development of these tracts is the cause of the disease. In others the fibres have been few in number and incompletely myelinized. This hypothesis has been urged by Marie, who limits the name spastic paraplegia to that group of the infantile cases in which there is no evidence of involvement of the brain—intellectual disturbances, epilepsy, etc., and it is in these cases that he believes the pyramidal tracts have remained undeveloped.

Symptoms.—At first nothing abnormal may be noticed about the child. In some instances there have been early and frequent convulsions; then at the age when the child should begin to walk it is noticed that the limbs are not used readily, and on examination a stiffness of the legs and arms is found. Even at the age of two the child may not be able to sit up, and often the head is not well supported by the neck muscles. The rigidity, as a rule, is more marked in the legs, and there is an adductor spasm. When supported on the feet, the child either rests on its toes and the inner surface of the feet, with the knees close together, or the legs may be crossed. The stiffness of the upper limbs varies. It may be scarcely noticeable or the rigidity may be as marked as in the legs. When the spastic condition affects the arms as well as the legs, we speak of the condition as diplegia; when the legs alone are involved, as paraplegia. There seems to be no sufficient reason for considering them separately. The spasticity is probably due to interruption of the cortico-spinal fibres which exercise an inhibitory influence on the cells of the anterior horns. Constant irregular movements of the arms are not uncommon. The child has great difficulty in grasping an object. The spasm and weakness may be more evident on one side than the other. The mental condition is, as a rule, defective and convulsive seizures are common.

Associated with the spastic paralysis are two allied conditions of considerable interest, characterized by spasm and disordered movements. A child with spastic diplegia may present, in an unusual degree, irregular movements of the muscles. In attempting to grasp an object the fingers may be thrown out in a stiff, spasmodic, irregular manner, or there may be constant irregular

movements of the shoulders, arms, and hands, with slight incoördination of the head. Cases of this description have been described as *chorea spastica*, and they may be difficult to separate from multiple sclerosis and from Friedreich's ataxia.

A still more remarkable condition is that of *bilateral athetosis*, in which there is a combination of spasm more or less marked with the most extraordinary bizarre movements of the muscles. The condition, as a rule, dates from infancy. The patient may not be able to walk. The head is turned from side to side; there are continual irregular movements of the face muscles, and the mouth is drawn and greatly distorted. The extremities are more or less rigid, particularly in extension. On the slightest attempt to move, often spontaneously, there are extraordinary movements of the arms and legs, particularly of the arms, somewhat like athetosis, though much more exaggerated. The patients are often unable to help themselves on account of these movements. The reflexes are increased. The mental condition is variable. The patient may be idiotic, but in 3 of the 6 cases which I have seen the patients were intelligent. Massalongo, who has carefully studied this condition, describes 3 cases in one family. I have collected 53 cases from the literature, 33 of which occurred in males and 20 in females.

Treatment.—Little can be done for these children when the symptoms are extreme. In the milder cases patient training may do much to better the mental state when feeble-mindedness accompanies the motor palsies. Exercises and massage should be given for the spastic muscles, and in many instances tenotomies and tendon transplantations may be helpful in improving the usefulness particularly of the lower extremities. Division of the posterior nerve roots is of great use in certain cases. On the view that most of these cases date back to an intracranial hæmorrhage during parturition, it is reasonable to suppose that an immediate operation with the removal of the cortical clot—for the effusion of blood is usually on the surface of the hemisphere—might ward off the disastrous consequences of compression on the infant's brain. Cases, with asphyxia and convulsions after difficult labors, have been operated upon soon after birth by Cushing and others, and cortical clots have been removed. In some cases there has been a complete restoration to health and the usual spastic sequels have not occurred.

3. HEREDITARY SPASTIC PARAPLEGIA

(*Hereditary Spastic Spinal Paralysis; Family form of Spastic Spinal Paralysis*)

It is a family affection and only occasionally are the ascendants affected. There are several forms:

1. The pure spastic paraplegia—Strümpell's type—in which two or more members of a family are attacked. Trunk, arms, and brain are not affected.

2. Mixed forms: (*a*) with features of multiple sclerosis as described by Cestan and Guillain; (*b*) amyotrophic lateral sclerosis type, with the added feature of atrophy; (*c*) forms resembling Friedreich's ataxia and the hereditary cerebellar ataxia; (*d*) forms resembling cerebral diplegia.

In a majority of the cases the disease begins in children between the sev-

enth and the fifteenth years. It may not occur until the twentieth year. Two, three, or four members of a family are affected. Beginning in the legs with characteristic spastic gait and all the features of an ordinary spinal paralysis, the disease may extend and affect the arms, or there are added the symptoms of multiple sclerosis or of one of the other above-named affections. Boys are more often affected than girls, 88 to 51, in the cases collected by Deléarde and Minet.

The pathology of the disease is still under discussion.

Amaurotic Family Idiocy (*Sachs' Disease*).—A remarkable form of infantile paralysis has been described by Sachs, Peterson, and Hirsch. The disease is one which involves the entire gray matter of the central nervous system. The symptoms as summarized by Sachs are: (1) Psychic disturbances that appear in early life (first or second year) and progress to total idiocy. (2) Paresis, and ultimately complete paralysis of the extremities, which may be either flaccid or spastic. (3) Increased, decreased, or normal tendon reflexes. (4) Partial, followed by total blindness (macular changes, with subsequent atrophy of the optic nerve). (5) Marasmus and death, usually before the second year. (6) Distinct familial type. Occasional symptoms are nystagmus, strabismus, hyperacusis, or impairment of hearing. The pathological changes are primitive type of the cerebral convolutions, macrogyria, degenerative changes in the large pyramidal cells, absence of the tangential fibres, and decrease of the fibres of the white matter. The blood-vessels are normal. There is also degeneration of the pyramidal columns of the cord. Of 27 cases collected by Sachs, 17 occurred in six families; all in Hebrews.

4. ERB'S SYPHILITIC SPINAL PARALYSIS

Erb has described a symptom group under the term syphilitic spinal paralysis, to which much attention has been given. The points upon which he lays stress are a very gradual onset with a development finally of the features of a spastic paresis; the tendon reflexes are greatly increased, but the muscular rigidity is slight in comparison with the exaggerated deep reflexes. There is rarely much pain, and the sensory disturbances are trivial, but there may be paræsthesia and the girdle sensation. The bladder and rectum are usually involved, and there is sexual failure or impotence. And, lastly, improvement is not infrequent. A majority of instances of spastic paralysis of adults not the result of slow compression of the cord are associated with syphilis and belong to this group.

Erb believes the lesion to be a special form of transverse myelitis, but perhaps it should be classed, with the system diseases, under the name toxic spastic spinal paralysis.

5. SECONDARY SPASTIC PARALYSIS

Following any lesion of the pyramidal tract there may be a spastic paralysis; thus, in a transverse lesion of the cord, whether the result of slow compression (as in caries), chronic myelitis, the pressure of tumor, chronic meningo-myelitis, or multiple sclerosis, degeneration takes place in the pyramidal tracts, below the point of disease. The legs soon become stiff and rigid, and the reflexes increase. Bastian has shown that in compression paraplegia if the

transverse lesion is complete, the limbs may be flaccid, without increase in the reflexes—*paraplégie flasque* of the French. The condition of the patient in these secondary forms varies very much. In chronic myelitis or in multiple sclerosis he may be able to walk about, but with a characteristic spastic gait. In the compression myelitis, in fracture, or in caries, there may be complete loss of power with rigidity.

It may be difficult or even impossible to distinguish these cases from those of primary spastic paralysis. Reliance is to be placed upon the associated symptoms; when these are absent no definite diagnosis as to the cause of the spastic paralysis can be given.

6. HYSTERICAL SPASTIC PARAPLEGIA

There is no spinal cord disease which may be so accurately mimicked as spastic paraplegia. In the hysterical form there is wasting, the sensory symptoms are not marked, the loss of power is not complete, and there is not that extensor spasm so characteristic of organic disease. The reflexes are, as a rule, increased. The knee-jerk is present, and there may be a well developed ankle clonus. Gowers calls attention to the fact that it is usually a spurious clonus, "due to a half-voluntary contraction in the calf muscles." A true clonus does occur, however, and there may be the greatest difficulty in determining whether or not the case is one of hysterical paraplegia. The hysterical contracture will be considered later.

C. SYSTEM DISEASES OF THE LOWER MOTOR SEGMENT

1. CHRONIC ANTERIOR POLIO-MYELITIS

(*Progressive Muscular Atrophy—Aran-Duchenne*)

This disease has been considered as one of the types making up the progressive (central) muscular atrophies. In certain rare cases the process is confined to the lower motor segment. They, however, differ so little clinically from many of the cases in which the pyramidal tracts are involved that it seems better to make no sharp distinction between them. The same may be said of chronic bulbar paralysis.

2. OPHTHALMOPLEGIA

This disease is at times due to a chronic degeneration of the nuclei of the motor nerves of the eyeballs, and so is a system disease of the lower motor segment. It is treated of in connection with the other ocular palsies for the sake of simplicity and because all ophthalmoplegias are not due to nuclear disease.

3. ACUTE POLIO-MYELITIS

(*Infantile Spinal Paralysis, Heine-Medin Disease*)

The epidemic form of this affection has been considered with the infectious diseases. The sporadic cases are very probably, although not surely, due to

the same infection. They present the same clinical picture and need no further consideration.

4. ACUTE AND SUBACUTE POLIO-MYELITIS IN ADULTS

An acute polio-myelitis in adults, the exact counterpart of the disease in children, is recognized. Many of the cases represent the sporadic form of polio-myelitis. A certain number of the cases described under this heading have been multiple neuritis; but the suddenness of onset, the rapid wasting, and the marked reaction of degeneration are thought by some to be distinguishing features. Multiple neuritis may, however, set in with rapidity; there may be great wasting and the reaction of degeneration is sometimes present. The time element alone may determine the true nature. Recovery in a case of extensive multiple paralysis from polio-myelitis will certainly be with loss of power in certain groups of muscles; whereas, in multiple neuritis the recovery, while slow, may be perfect.

The subacute form, the *paralysie générale spinale antérieure subaiguë* of Duchenne, is in all probability a peripheral palsy. The paralysis usually begins in the legs with atrophy of the muscles, then the arms are involved, but not the face. Sensation, as a rule, is not involved.

5. ACUTE ASCENDING (LANDRY'S) PARALYSIS

Definition.—An ascending flaccid paralysis, beginning in the legs, rapidly extending to the trunk and arms, and finally involving the muscles of respiration. Sensation and electrical reactions are normal, and there is retention of sphincter control.

Etiology and Pathology.—The disease occurs most commonly in males between the twentieth and thirtieth years. It has sometimes followed the specific fevers, As already mentioned, there is a form of the epidemic polio-myelitis which has an acute course and a clinical picture similar to Landry's paralysis, but it is not likely, as has been suggested, that this disease always represents the sporadic variety. Many of the common pathogenic organisms may, especially in patients debilitated by disease, give rise to symptoms of acute ascending paralysis. Thus, the typhoid bacillus may produce clinically an acute ascending paralysis. The most careful studies have not solved the problem of this remarkable disease. There are two views: First, that it is a peripheral neuritis. Spiller in a rapidly fatal case found destructive changes in the peripheral nerves and corresponding alterations in the cell bodies of the ventral horns. He suggests that the toxic agent acts on the lower motor neurones as a whole, and that possibly the reason why no lesions were found in some of the cases is that the more delicate histological methods were not used. Buzzard has isolated a micrococcus (*M. tetragenus*) in pure culture in one case, and found the organism in large numbers in the tissues outside the spinal dura. Secondly, that it is a functional disorder without a recognizable anatomical basis.

Symptoms.—Weakness of the legs, gradually progressing, often with tolerable rapidity, is the first symptom. In some cases within a few hours the paralysis of the legs becomes complete. The muscles of the trunk are next

affected, and within a few days, or even less in more acute cases, the arms are also involved. The neck muscles are next attacked, and finally the muscles of respiration, deglutition, and articulation. The reflexes are lost, but the muscles neither waste nor show electrical changes. The sensory symptoms are variable; in some cases tingling, numbness, and hyperæsthesia have been present. In the more characteristic cases sensation is intact and the sphincters are uninvolved. Enlargement of the spleen, which occurred in the only two cases in my wards, has been noticed in several other cases. The course of the disease is variable. It may prove fatal in less than two days. Other cases persist for a week or for two weeks. In a large proportion of the cases the disease is fatal. One patient was kept alive for 41 days by artificial respiration (C. L. Greene).

Diagnosis.—The diagnosis is difficult, particularly from certain forms of multiple neuritis, and if we include in Landry's paralysis the cases in which sensation is involved distinction between the two affections is impossible. We apparently have to recognize the existence of a rapidly advancing motor paralysis without involvement of the sphincters, without wasting or electrical changes in the muscles, without trophic lesions, and without fever—features sufficient to distinguish it from either the acute central myelitis or the polio-myelitis anterior. It is doubtful, however, whether these characters always suffice to enable us to differentiate the cases of multiple neuritis. The cases of acute polio-myelitis with the picture of an acute ascending paralysis should not be difficult to recognize during the progress of an epidemic.

IV. COMBINED SYSTEM DISEASES

When the disease is not confined within the limits of either the afferent or efferent systems, but affects both, it is known as a *combined system disease*. Some authors contend that the diseases usually classed under this head are not really system diseases, but are diffuse processes. This is the view taken by Leyden and Goldscheider, who limit the term system disease to locomotor ataxia and progressive muscular atrophy.

In certain cases of locomotor ataxia which have run a fairly typical course there may be found after death, besides the anatomical picture corresponding to this disease, a moderate degeneration of the pyramidal tracts and of the ventral horns. In progressive muscular atrophy, on the other hand, there may be degeneration in the dorsal columns. During life these secondary involvements of other systems may or may not be accompanied by demonstrable symptoms, and when such do occur they make their appearance late in the disease.

There is another group of cases in which from the very first the symptoms point to an involvement of both the afferent and efferent systems, and it is to these that the term primary combined system disease is usually limited.

1. ATAXIC PARAPLEGIA

This name is applied by Gowers to a disease characterized clinically by a combination of ataxia and spastic paraplegia, and anatomically by involvement of the dorsal and lateral columns.

The disease is most common in middle aged males. Exposure to cold and traumatism have been occasional antecedents. In striking contrast to ordinary tabes a history of syphilis is rarely to be obtained.

The anatomical features are a sclerosis of the dorsal columns, which is not more marked in the lumbar region and not specially localized in the root zone of the cuneate fasciculi. The involvement of the lateral columns is diffuse, not always limited to the pyramidal tracts, and there may be an annular sclerosis. Marie believes that in many cases the distribution of the sclerosis is due to the arterial supply and not to a true systematic degeneration, the vessels involved being branches of the dorsal spinal artery.

The *symptoms* are well defined. The patient complains of a tired feeling in the legs, not often of actual pain. The sensory symptoms of true tabes are absent. An unsteadiness in the gait gradually comes on with progressive weakness. The reflexes are increased from the outset, and there may be well marked ankle clonus. Rigidity of the legs comes on slowly, but it is rarely extreme as in the uncomplicated cases of lateral sclerosis. From the start incoördination is a well characterized feature, and the difficulty of walking in the dark, or swaying when the eyes are closed, may, as in true tabes, be the first symptom to attract attention. In walking the patient uses a stick, keeps the eyes fixed on the ground, the legs far apart, but the stamping gait, with elevation and sudden descent of the feet, is not often seen. The incoordination may extend to the arms. Sensory symptoms are rare, but Gowers calls attention to a dull, aching pain in the sacral region. The sphincters usually become involved. Eye symptoms are rare. Late in the disease mental symptoms may occur, similar to those of general paresis.

In well marked cases the *diagnosis* is easy. The combination of marked incoördination with retention of the reflexes and more or less spasm are characteristic features. The absence of ocular and sensory symptoms is an important point.

2. PRIMARY COMBINED SCLEROSIS (PUTNAM)

The studies of J. J. Putnam, Dana, Bastianelli, Risien Russell, Collier, and Batten have separated from among the lesions of the cord a fairly well defined disease, characterized anatomically by a diffuse degeneration, often in discrete patches. The dorsal and lateral columns are constantly involved, chiefly in the thoracic and cervical regions. The nerve roots and the gray matter show no changes. The lesions have the "appearance of a non-systemic primary neurone degeneration, not dependent upon antecedent inflammation" (E. W. Taylor).

Of Putnam's 50 cases, 31 were women, all but 5 above thirty years old. A majority of the patients were of small stature and slender frame, and in many there had been a general lack of vigor and a chronic pallor and debility; 7 presented profound anæmia. There was no luetic history. The relation of this group to anæmia is interesting. Russell, Batten, and Collier make three groups: (1) cases of profound anæmia (and one may add of cachexia), in which during life no symptoms were present, but in which there were found combined scleroses of the cord post mortem; (2) cases of progressive pernicious anæmia, in which spinal symptoms have occurred; (3) cases of chronic

sclerosis of the cord, in which there occurs, as a secondary feature, a severe anæmia.

The *symptoms* are both sensory and motor. The onset is usually with numbness in the extremities, progressive loss of strength, and emaciation. Paraplegia gradually develops, before which there have been, as a rule, spastic symptoms with exaggerated knee-jerk. The arms are affected less than the legs. Mental symptoms suggestive of dementia paralytica may occur toward the close.

3. HEREDITARY ATAXIA

(*Friedreich's Ataxia*)

In 1861 Friedreich reported 6 cases of a form of hereditary ataxia, and the affection has usually gone by his name. Unfortunately, *paramyoclonus multiplex* is also called Friedreich's disease; so it is best, if his name is used in connection with this affection, to term it Friedreich's ataxia. It is a very different disease in many respects from ordinary tabes. It may or may not be hereditary. It is really a family disease, several brothers and sisters being, as a rule, affected. The 143 cases analyzed by Griffith occurred in 71 unrelated families. In his series inheritance of the disease itself occurred in only 33 cases. Various influences in the parents have been noted; alcoholism in only 7 cases. Syphilis has rarely been present. Of the 143 cases, 86 were males and 57 females. The disease sets in early in life, and in Griffith's series 15 occurred before the age of two years, 39 before the sixth year, 45 between the sixth and tenth, 20 between the eleventh and fifteenth, 18 between the sixteenth and twentieth, and 5 between the twentieth and twenty-fifth years.

The *morbid anatomy* shows an extensive sclerosis of the dorsal and lateral columns of the spinal cord. The periphery and the cerebellar tracts are usually involved. The observations of Dejerine and Letulle are of special interest, since they seem to indicate that the change in this disease is a neurogliar (ectodermal) sclerosis, differing entirely from the ordinary spinal sclerosis. According to this view, Friedreich's disease is a gliosis of the dorsal columns due to developmental errors; but the question is still unsettled.

Symptoms.—The ataxia differs somewhat from the ordinary form. The incoördination begins in the legs, but the gait is peculiar. It is swaying, irregular, and more like that of a drunken man. There is not the characteristic stamping gait of the true tabes. Romberg's symptom may or may not be present. The ataxia of the arms occurs early and is very marked; the movements are almost choreiform, irregular, and somewhat swaying. In making any voluntary movement the action is overdone, the prehension is clawlike, and the fingers may be spread or overextended just before grasping an object. The hand frequently moves about an object for a moment and then suddenly pounces upon it. There are irregular, swaying movements of the head and shoulders, some of which are choreiform. There is present in many cases what is known as static ataxia, that is to say, ataxia of quiet action. It occurs when the body is held erect or when a limb is extended—irregular, oscillating movements of the head and body or of the extended limb.

Sensory symptoms are not usually present. The deep reflexes are lost early in the disease, and, next to the ataxia, this is the most constant and important

symptom. The skin reflexes are usually normal, and the pupillary reflex to light is practically never affected.

Nystagmus is a characteristic symptom. Atrophy of the optic nerve rarely occurs. A striking feature is early deformity of the feet. There is talipes equinus, and the patient walks on the outer edge of the feet. The big toe is flexed dorsally on the first phalanx. Scoliosis is very common.

Trophic lesions are rare. As the disease advances paralysis comes on and may ultimately be complete. Some of the patients never walk.

Disturbance of speech is common. It is usually slow and scanning; the expression is often dull; the mental power is, as a rule, maintained, but late in the disease becomes impaired.

Diagnosis.—The diagnosis of the disease is not difficult when several members of a family are affected. The onset in childhood, the curious form of incoördination, the loss of knee-jerks, the early talipes equinus, the position of the great toe, the scoliosis, the nystagmus, and scanning speech make up an unmistakable picture. The disease is often confounded with chorea, with the ordinary form of which it has nothing in common. With hereditary chorea it has certain similarities, but usually this disease does not set in until after the thirtieth year.

The affection lasts for many years and is incurable. Care should be taken to prevent contractures.

Cerebellar Type

There is a form of hereditary ataxia, described by Marie as *cerebellar heredo-ataxia*, which starts later in life, after the age of twenty, with disability in the legs, but the gait is less ataxic than "groggy." The knee-jerks are retained, and a spastic condition of the legs ultimately develops. There is no scoliosis, nor does club-foot develop. Sanger Brown's cases, 25 in one family, and J. H. Neff's, 13, appear to belong to this type. The cerebellum has been found atrophied in 2 cases.

4. PROGRESSIVE INTERSTITIAL HYPERTROPHIC NEURITIS OF INFANTS

Under this imposing title Dejerine and Sottas described a rare and interesting affection. It is a family disease, and begins in early life. The symptoms are those typical of locomotor ataxia, to which is added progressive muscular atrophy, with involvement of the face and a hypertrophy and hardening of the peripheral nerves. As the name indicates, it is an interstitial hypertrophic neuritis with secondary involvement of the dorsal columns of the cord. This disease has been associated with progressive neural muscular atrophy, but Dejerine has shown that it is quite distinct.

5. TOXIC COMBINED SCLEROSIS

Certain poisons cause changes in the lateral and dorsal columns of the cord that resemble those of the combined system diseases. They have been demonstrated in pellagra and in ergotism. In pernicious anæmia and many chronic wasting diseases these scleroses occur, and are believed to be due to the action of poisons produced within the system.

C. DIFFUSE DISEASES OF THE NERVOUS SYSTEM

I. AFFECTIONS OF THE MENINGES

DISEASES OF THE DURA MATER

(*Pachymeningitis*)

Pachymeningitis Externa.—CEREBRAL.—Hæmorrhage often occurs as a result of fracture. Inflammation of the external layer of the dura is rare. Caries of the bone, either extension from middle-ear disease or due to syphilis, is the principal cause. In the syphilitic cases there may be a great thickening of the inner table and a large collection of pus between the dura and the bone.

Occasionally the pus is infiltrated between the two layers of the dura mater or may extend through and cause a dura-arachnitis.

The symptoms of external pachymeningitis are indefinite. In the syphilitic cases there may be a small sinus communicating with the exterior. Compression symptoms may occur with or without paralysis.

SPINAL.—An acute form may occur in syphilitic affections of the bones, in tumors, and in aneurism. The symptoms are those of a compression of the cord. A chronic form is much more common, and is a constant accompaniment of tuberculous caries of the spine. The internal surface of the dura may be smooth, while the external is rough and covered with caseous masses. The entire dura may be surrounded, or the process may be confined to the ventral surface.

Pachymeningitis Interna —This occurs in three forms: (1) Pseudo-membranous, (2) purulent, and (3) hæmorrhagic. The first two are unimportant. Pseudo-membranous inflammation of the lining membrane of the dura is not usually recognized, but a most characteristic example of it came under my observation as a secondary process in pneumonia. Purulent pachymeningitis may follow an injury, but is more commonly the result of extension from inflammation of the pia. It is remarkable how rarely pus is found between the dura and arachnoid membranes.

HÆMORRHAGIC PACHYMENINGITIS

~~(*Hæmatoma of the Dura Mater*)~~

Cerebral Form.—This remarkable condition, first described by Virchow, is very rare in general medical practice. During ten years no ~~instance of it came under my observation~~ at the Montreal General Hospital. On the other hand, in the post-mortem room of the Philadelphia Hospital, which received material from a large almshouse and asylum, the cases were not uncommon, and within three months ~~I saw~~ four characteristic examples, three of which came from the medical wards. The frequency ~~of the condition~~ in asylum work may be gathered from the fact that in 1,185 post mortems at the Government Hospital for the Insane, Washington, to June 30, 1897, there were 197 cases with "a true neo-membrane of internal pachymeningitis" (Blackburn). Of these cases, 45 were chronic dementia, 37 were general paresis, 30 senile de-

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Spinal Form.—The spinal *pachymeningitis interna*, described by Charcot and Joffroy, involves chiefly the cervical region (*P. cervicalis hypertrophica*). The space between the cord and the dura is occupied by a firm, concentrically arranged, fibrinous growth, which is seen to have developed within, not outside of, the dura mater. It is a condition anatomically identical with the hæmorrhagic pachymeningitis interna of the brain. The

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mentia, 28 chronic mania, 28 chronic melancholia, 22 chronic epileptic insanity, 6 acute mania, and 1 case imbecility. Forty-two of the cases were in persons over seventy years of age.

It has also been found in profound anæmia and other diseases of the blood and of the blood vessels, and is said to have followed certain of the acute fevers. Herter called attention to the not infrequent occurrence of the lesion in badly nourished cachectic children.

The morbid anatomy is interesting. Virchow's view that the delicate vascular membrane precedes the hæmorrhage is undoubtedly correct. Practically we see one of three conditions in these cases: (*a*) subdural vascular membranes, often of extreme delicacy; (*b*) simple subdural hæmorrhage; (*c*) a combination of the two, vascular membrane and blood clot. Certainly the vascular membrane may exist without a trace of hæmorrhage—simply a fibrous sheet of varying thickness, permeated with large vessels, which may form beautiful arborescent tufts. On the other hand, there are instances in which the subdural hæmorrhage is found alone, but it is possible that in some of these at least the hæmorrhage may have destroyed all trace of the vascular membrane. In some cases a series of laminated clots are found, forming a layer from 3 to 5 mm. in thickness. Cysts may occur within this membrane. The source of the hæmorrhage is probably the dural vessels. Huguenin and others hold that the bleeding comes from the vessels of the pia mater, but certainly in the early stage of the condition there is no evidence of this; on the other hand, the highly vascular subdural membrane may be seen covered with the thinnest possible sheeting of clot, which has evidently come from the dura. The subdural hæmorrhage is usually associated with atrophy of the convolutions, and it is held that this is one reason why it is so common in the insane, especially in dementia paralytica and dementia senilis. We meet with the condition also in phthisis and various cachectic conditions in which the cerebral wasting is as common and almost as marked as in cases of insanity. König found in 135 cases of hæmorrhagic pachymeningitis from the Berlin Pathological Institute that 23 per cent. accompanied phthisis. Atrophy, however, may not be the only factor.

The symptoms are indefinite, or there may be none at all, especially when the hæmorrhages are small or have occurred very gradually, and the diagnosis can not be made with certainty. Headache has been a prominent symptom in some cases, and when the condition exists on one side there may be hemiplegia. The most helpful symptoms for diagnosis, indicating that the hæmorrhage in an apoplectic attack is meningeal, are (1) those referable to increased intracranial pressure (slowing and irregularity of the pulse, vomiting, coma, contracted pupils, reacting to light slowly or not at all) and (2) paresis and paralysis, gradually increasing in extent, accompanied by symptoms which point to a *cortical* origin. Extensive bilateral disease may, however, exist without any symptoms whatever.

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cord is usually compressed; the central canal may be dilated—hydromyelus—and there are secondary degenerations. The nerve roots are involved in the growth and are damaged and compressed. The extent is variable. It may be limited to one segment, but more commonly involves a considerable portion of the cervical enlargement. The disease is chronic, and in some cases presents a characteristic group of symptoms. There are intense neuralgic pains in the course of the nerves whose roots are involved. They are chiefly in the arms and in the cervical region, and vary greatly in intensity. There may be hyperæsthesia with numbness and tingling; atrophic changes may develop, and there may be areas of anæsthesia. Gradually motor disturbances appear; the arms become weak and the muscles atrophied, particularly in certain groups, as the flexors of the hand. The extensors, on the other hand, remain intact, so that the condition of claw-hand is gradually produced. The grade of the atrophy depends much upon the extent of involvement of the cervical nerve roots, and in many cases the atrophy of the muscles of the shoulders and arms becomes extreme. The condition is one of cervical paraplegia, with contractures, flexion of the wrist, and typical *main en griffe*. Usually before the arms are greatly atrophied there are the symptoms of what the French writers term the second stage—namely, involvement of the lower extremities and the gradual production of a spastic paraplegia, which may develop several months after the onset of the disease, and is due to secondary changes in the cord.

The disease runs a chronic course, lasting, perhaps, two or more years. In a few instances, in which symptoms pointed definitely to this condition, recovery has taken place. The disease is to be distinguished from amyotrophic lateral sclerosis, syringomyelia, and tumors. From the first it is separated by the marked severity of the initial pains in the neck and arms; from the second by the absence of the sensory changes characteristic of syringomyelia. From certain tumors it is very difficult to distinguish; in fact, the fibrinous layers form a tumor around the cord.

The condition known as *hæmatoma* of the dura mater may occur at any part of the cord, or, in its slow, progressive form—pachymeningitis hæmorrhagica interna—may be limited to the cervical region and produce the symptoms just mentioned. It is sometimes extensive, and may coexist with a similar condition of the cerebral dura. Cysts may occur filled with hæmorrhagic contents.

DISEASES OF THE PIA MATER

Acute Cerebro-spinal Leptomeningitis

Etiology.—Under cerebro-spinal fever and tuberculosis the two most important forms of meningitis have been described. Other conditions with which meningitis is associated are: (1) *The acute fevers,* more particularly pneumonia, erysipelas, and septicæmia; less frequently small-pox, typhoid fever, scarlet fever, measles, influenza, etc. (2) *Injury or disease of the bones of the skull.* In this group by far the most frequent cause is necrosis of the petrous portion of the temporal bone in chronic otitis. (3) *Extension from disease of the nose.* Meningitis has followed perforation of the skull in sounding the frontal sinuses, suppurative disease of these sinuses, and necroses of the cribriform plate. As mentioned under cerebro-spinal fever, the infection is thought

to be possible through the nose. (4) As a *terminal infection* in chronic nephritis, arterio-sclerosis, heart disease, gout, and the wasting diseases of children.

The following etiological table of the chief acute forms of meningitis may be useful to the student:

Acute Leptomeningitis.	Primary.	1. Of cerebro-spinal fever.	(*a*) Sporadic. (*b*) Epidemic.	Diplococcus intracellularis.
		2. Pneumococcic.	Meninges involved alone or in a general pneumococcus infection.	Pneumococcus.
	Secondary.	1. Tuberculous		Bacillus tuberculosis.
		2. Pneumococcic.	(*a*) Secondary to pneumonia, endocarditis, etc.	Pneumococcus.
			(*b*) Secondary to disease or injury of cranium or its fossæ.	
		3. Pyogenic.	(*a*) Following local disease of cranium or a local infection elsewhere.	Various forms of staphylococci and streptococci.
			(*b*) Terminal infection in various chronic maladies.	
		4. Miscellaneous acute infections.	In typhoid fever, influenza, diphtheria, gonorrhœa, anthrax, actinomycosis, and other acute diseases.	Typhoid bacillus, influenza bacillus, diphtheria bacillus, gonococcus, etc.

Morbid Anatomy.—The basal or cortical meninges may be chiefly attacked. The degree of involvement of the spinal meninges varies. In the form associated with pneumonia and ulcerative endocarditis the disease is bilateral and usually limited to the cortex. In extension from disease of the ear it is often unilateral and may be accompanied with abscess or with thrombosis of the sinuses. In the non-tuberculous form in children, in the meningitis of chronic Bright's disease, and in cachectic conditions the base is usually involved. In the cases secondary to pneumonia the effusion beneath the arachnoid may be very thick and purulent, completely hiding the convolutions. The ventricles also may be involved, though in these simple forms they rarely present the distention and softening which are so frequent in the tuberculous meningitis. For a more detailed description the student is referred to the sections on cerebro-spinal fever and tuberculous meningitis.

Symptoms.—The clinical features of meningitis have already been described at length in the diseases just ~~referred to, and I shall here give a general summary.~~ Cortical meningitis is not to be recognized by any symptoms or set of symptoms from a condition which may be produced by the poison of many of the specific fevers. In the cases of so-called cerebral pneumonia, unless the base is involved and the nerves affected, the disease is unrecognizable, since identical symptoms may be produced by intense engorgement of the meninges. In typhoid fever, in which meningitis is very rare, the twitchings, spasms, and retractions of the neck are almost invariably associated with cerebro-spinal congestion, not with meningitis. Actual meningitis does, however, occur in typhoid fever, and typhoid bacilli may be present in the exudate.

A knowledge of the etiology gives a very important clew. Thus, in middle-ear disease the development of high fever, delirium, vomiting, convulsions, and retraction of the head and neck would be extremely suggestive of meningitis or abscess. Headache, which may be severe and continuous, is the most common symptom. While the patient remains conscious this is usually the chief complaint, and even when semicomatose he may continue to groan and to

place his hand on his head. In the fevers, particularly in pneumonia, there may be no complaint of headache. Delirium is frequently early, and is most marked when the fever is high. Convulsions are less common in simple than in tuberculous meningitis. They were not present in a single instance in the cases which I have seen in pneumonia, ulcerative endocarditis, or septicæmia. In the simple meningitis of children they may occur. Epileptiform attacks which come and go are highly characteristic of direct irritation of the cortex. Rigidity and spasm or twitchings of the muscles are more common. Stiffness and retraction of the muscles of the neck are important symptoms; but they are by no means constant, and are most frequent when the inflammation is extensive on the meninges of the cervical cord. There may be trismus, gritting of the teeth, or spastic contraction of the abdominal muscles. Vomiting is a common symptom in the early stages, particularly in basilar meningitis. Constipation is usually present. In the late stages the urine and fæces may be passed involuntarily. Optic neuritis is rare in the meningitis of the cortex, but is not uncommon when the base is involved. Leube lays stress on the hyperæsthesia of the skin and muscles, especially of the muscles of the neck and calves.

Important symptoms are due to lesions of the nerves at the base. Strabismus or ptosis may occur. The facial nerve may be involved, producing slight paralysis, or there may be damage to the fifth nerve, producing anæsthesia and, if the Gasserian ganglion is affected, trophic changes in the cornea. The pupils are at first contracted, subsequently dilated, and perhaps unequal. The reflexes in the extremities are often accentuated at the beginning of the disease; later they are diminished or entirely abolished. Herpes is common, particularly in the epidemic form.

Fever is present, moderate in grade, rarely rising above 103°. In the non-tuberculous leptomeningitis of debilitated children and in Bright's disease there may be little or no fever. The pulse may be increased in frequency at first, though this is unusual. One of the striking features of the disease is the slowness of the pulse in relation to the temperature, even in the early stages. Subsequently it may be irregular and still slower. The very rapid emaciation which often occurs is doubtless to be referred to a disturbance of the cerebral influence upon metabolism. Kernig's sign has been described under cerebro-spinal fever. There may be a concomitant reflex of one leg when passive flexion is made of the other (Brudzinski's sign); and when the neck is bent forward there is flexion of the legs both at the knees and hips. Lumbar puncture is exceedingly valuable for diagnosis. It may be that a turbid fluid indicates an acute non-tuberculous meningitis. At first the fluid may be only opalescent. A close relationship exists between the severity of the symptoms, the height of the pyrexia, and the degree of turbidity (Connal). As a rule a preponderance of polynuclear leucocytes is present with the meningococcus or the pyogenic organisms; a mononuclear exudate is characteristic of tuberculosis. It is to be remembered that in tuberculous meningitis the fluid is usually clear; in only one of 69 cases was it opalescent (Connal).

Treatment.—There are no remedies which in any way control the course of acute meningitis. An ice-bag should be applied to the head. and, if the subject is young and full blooded, general or local depletion may be practised. Absolute rest and quiet should be enjoined. When disease of the ear is

Normal in children. min. 0.054% max. 0.139% much like same as for blood sugar.

Glucose in cereb. spin. fluid in acute Meningitis.
sugar in blood in such infectious diseases.

Meningococcus cases. Sugar disappears more or less completely.
reappears about 5th day in favorable cases & is reduced
again when relapse comes on
& is not reduced when disturbance in nucleus takes place

Persistently low sugar content. is an unfavorable sign

Test. 3 drops of Fehling's soln. added to 2 cc. of centrifuged or filtered cereb. fluid. Boil. If distinct & prompt precipitation - positive.
If blue disappears without ppt. means reduction of sugar - but not entirely absent.

present, a surgeon should be early called in consultation, and if there are symptoms of meningo-encephalitis which can in any way be localized trephining should be practised. An occasional saline purge will do more to relieve the congestion than blisters and local depletion. The warm baths, as recommended by Aufrecht and described under cerebro-spinal fever, should be given every three hours. It is possible that recovery may follow in the primary pneumococcus form (Netter). If counter-irritation is deemed essential, the thermo-cautery may be lightly applied to the back of the neck. Large doses of the perchloride of iron, iodide of potassium, and mercury are recommended by some authors. Hexamethylenamine in doses of 60 grains (4 gm.) daily may be tried, as Crowe has shown that it is excreted in the cerebro-spinal fluid and controls the growth of organisms in the meninges.

The application of an ice-cap, attention to the bowels and stomach, and keeping the fever within moderate limits by sponging are the necessary measures in a disease recognized as almost invariably fatal, in which also the cases of recovery are extremely doubtful. Lumbar puncture may be used as a therapeutic measure.

The **posterior basic meningitis** of Gee, Lees, and Bartow is the sporadic form of cerebro-spinal fever and has been already described.

Meningism.—Sometimes spoken of as the syndrome of Dupré, this is a condition in which there are symptoms of meningitis, but post mortem the characteristic pathological changes are not present. It is practically the condition described formerly as meningeal irritation, and is seen most frequently in the acute fevers of children, particularly in pneumonia and typhoid fever, sometimes in alcoholism and in middle-ear disease.

Chronic Leptomeningitis.—This is rarely seen apart from syphilis or tuberculosis, in which the meningitis is associated with the growth of the granulomata in the meninges and about the vessels. The symptoms in such cases are extremely variable, depending entirely upon the situation of the growth. The epidemic meningitis may run a very chronic course, but of all forms the posterior basic may be the most protracted, as cases have been described with a duration of a year or more. Quincke's *meningitis serosa* is considered with hydrocephalus.

II. SCLEROSES OF THE BRAIN

General Remarks.—The supporting tissue of the central nervous system is the neuroglia, derived from the ectoderm, with distinct morphological and chemical characters. The meninges are composed of true connective tissue derived from the mesoderm, a little of which enters the brain and cord with the blood-vessels. The neuroglia plays the chief part in pathological processes within the central nervous system, but changes in the connective tissue elements may also be important. A convenient division of the cerebro-spinal scleroses is into degenerative, inflammatory, and developmental forms.

The *degenerative scleroses* comprise the largest and most important subdivision, in which provisionally the following groups may be made: (*a*) The common secondary degeneration which follows when nerve fibres are cut off from their trophic centres; (*b*) toxic forms, among which may be placed the scleroses from lead and ergot, and, most important of all, the scleroses of the

dorsal columns, due in such a large proportion of cases to the virus of syphilis; (*c*) the sclerosis associated with change in the smaller arteries and capillaries, which is met with as a senile process in the convolutions.

The *inflammatory scleroses* embrace a less important and less extensive group, comprising secondary forms which follow irritative inflammation about tumors, foreign bodies, hæmorrhages, and abscess. Possibly a similar change may follow the primary, acute encephalitis, which Strümpell holds is the initial lesion in the cortical sclerosis which is so commonly found post mortem in infantile hemiplegia.

The *developmental scleroses* are believed to be of a purely neurogliar character, and embrace the new growth about the central canal in syringomyelia and, according to recent French writers, the sclerosis of the dorsal columns in Friedreich's ataxia.

Anatomically we meet with the following varieties:

Miliary sclerosis is a term which has been applied to several different conditions. Gowers mentions a case in which there were grayish red spots at the junction of the white and gray matters, and in which the neuroglia was increased. There is also a condition in which, on the surface of the convolutions, there are small nodular projections, varying from a half to five or more millimetres in diameter.

Diffuse sclerosis, which may involve an entire hemisphere, or a single lobe, in which case the term *sclérose lobaire* has been applied to it by the French. It is not an important condition in general medical practice, but occurs most frequently in idiots and imbeciles. In extensive cortical sclerosis of one hemisphere the ventricle is usually dilated. The symptoms of this condition depend upon the region affected. There may be a considerable extent of sclerosis without symptoms or without much mental impairment. In a majority of cases there is hemiplegia or diplegia with imbecility or idiocy.

Tuberous Sclerosis.—In this remarkable form, which is also known as hypertrophic sclerosis, there are on the convolutions areas, projecting beyond the surface, of an opaque white color and exceedingly firm. The sclerosis may not disturb the symmetry of the convolution, but simply cause a great enlargement, increase in the density, and a change in the color.

These three forms are not of much practical interest except in asylum and institution work. The fourth variety forms a well characterized disease of considerable importance, namely, multiple sclerosis.

Multiple (Insular: Disseminated) Sclerosis (*Sclérose en plaques.*)—Definition.—A chronic affection of the brain and cord, characterized by localized areas in which the nerve elements are more or less replaced by neuroglia. This may occur in the brain or cord alone, more commonly in both.

Etiology.—The cause is unknown. Kahler, Marie, and others assign great importance to the infectious diseases, particularly scarlet fever. Injury has occasionally preceded the onset. It is most common in young persons and in females. Several members in a family may be attacked.

Morbid Anatomy.—The sclerotic areas are widely distributed through the brain and cord, and cases limited to either part alone are almost unknown. The grayish red areas are scattered indifferently through the white and gray matter (E. W. Taylor). The patches are most abundant in the

neighborhood of the ventricles, and in the pons, cerebellum, basal ganglia, and the medulla. The cord may be only slightly involved or there may be very many areas throughout its length. The cervical region is apt to be most affected. The nerve roots and the branches of the cauda equina are often attacked. Histologically in the sclerosed patches there is a degeneration of the medullary sheaths, with the persistence for some time of the axis-cylinders. These naked axis-cylinders are thought by some to be new formed nerve fibres. Accompanying this there is marked proliferation of the neuroglia, the fibres of which are denser and firmer. Secondary degeneration, although relatively slight, does occur.

Symptoms.—The onset is slow and the disease is chronic. Feebleness of the legs with irregular pains and stiffness are among the early symptoms. Indeed, the clinical picture may be that of spastic paraplegia, with great increase in the reflexes. The following are the most important features:

(*a*) *Volitional Tremor or So-called Intention Tremor.*—There is no paralysis of the arms, but on attempting to pick up an object there is trembling or rapid oscillation. A patient may be unable to lift even a glass of water to the mouth. The tremor may be marked in the legs, and in the head, which shakes as he walks. When the patient is recumbent the muscles may be perfectly quiet. On attempting to raise the head from the pillow, trembling at once comes on. (*b*) *Scanning Speech.*—The words are pronounced slowly and separately, or the individual syllables may be accentuated. This staccato or syllabic utterance is a common feature. (*c*) *Nystagmus*, a rapid oscillatory movement of both eyes, is more common in multiple sclerosis than any other affection of the nervous system.

Sensation is unaffected in a majority of the cases. Optic atrophy may occur early, but is usually partial, rarely leading to complete blindness. The sphincters, as a rule, are unaffected until the last stages. Mental debility is not uncommon. Remarkable remissions occur in the course of the disease, in which for a time all the symptoms may improve. Vertigo is common, and there may be sudden apoplectiform attacks, such as occur in general paresis. The presence of the extensor plantar reflex (Babinski sign) and the absence of the abdominal reflexes are common.

The symptoms, on the whole, are extraordinarily variable, corresponding to the very irregular distribution of the nodules.

Diagnosis.—The diagnosis in well marked cases is easy. Volitional tremor, scanning speech, and nystagmus form a characteristic symptom-group. With this there is usually more or less spastic weakness of the legs. Paralysis agitans, certain cases of general paresis, and occasionally hysteria may simulate the disease very closely. If the case is not seen until near the end the diagnosis may be impossible. Buzzard holds that of all organic diseases of the nervous system disseminated sclerosis in its early stages is that which is most commonly taken for hysteria. The points to be relied upon in the differentiation are, in order of importance, optic atrophy, the nystagmus, the bladder disturbances, when present, and the volitional tremor. The tremor in hysteria is not volitional. Unilateral cases are recorded.

Pseudo-sclerosis—the Westphall-Strümpell disease—is a rare condition simulating multiple sclerosis and not often distinguished from it during life. Mental changes are more pronounced, the tremor is more exaggerated, the

nystagmus not always present, and the gait more ataxic. It sets in earlier, sometimes in the first decade, and in a majority of the cases no lesions have been found post mortem.

The PROGNOSIS is unfavorable. Ultimately, the patient, if not carried off by some intercurrent affection, becomes bedridden.

TREATMENT.—No known treatment has any influence on the progress of sclerosis of the brain. Neither the iodides nor mercury have the slightest effect, but a prolonged course of nitrate of silver or arsenic may be tried. The X-rays have been used with success (Raymond).

D. DIFFUSE AND FOCAL DISEASES OF THE SPINAL CORD

I. TOPICAL DIAGNOSIS

From the symptoms presented by a spinal cord lesion it is possible to determine more or less accurately not only the segmental level but also the transverse extent of the segmental involvement. The effects of an injury or of disease may be circumscribed and involve the gray matter of the segment or the tracts running through it; it may be more extensive and involve the cord in a given level in its entire transverse extent; finally, there are cases in which only one lateral half of the cord is implicated. It is well for the student to have a definite routine to follow in making his examinations, for each factor may be helpful in determining the site and character of the lesion. Some of the more important points to observe are the following: (1) *subjective sensations*, particularly the character and seat of pain, if any be present, such as the radiating pains of dorsal root compression; (2) the patient's *attitude*, as the position of the arms in cervical lesions, the character of the respiration, whether diaphragmatic, etc.; (3) *motor symptoms*, the groups of paralyzed muscles and their electrical reaction; (4) the *sensory symptoms*, including tests for tactual, thermic, and dolorous impressions, for muscle sense, bone sensation, etc.; (5) the condition of the *reflexes*, both the tendon and the skin reflexes as well as those for the pupil, the bladder and rectum, etc.; (6) the surface temperature and condition of moisture or dryness of the skin, which gives an indication of *vaso-motor* paralysis. The table on pages 898-900 and the figures on pages 906 and 907 will be useful while making an examination.

Focal Lesions.—We have seen that a lesion involving a definite part of the gray matter of the spinal cord, owing to destruction of the cell bodies of the lower motor neurones and consequent degeneration of their axis-cylinder processes, is accompanied by a loss of power to perform certain definite movements. Thus a disease, such as anterior poliomyelitis, may give as its only symptom a characteristic flaccid paralysis, and the seat of the lesion is revealed by the muscles involved. If from injury or disease a lesion involves more than the gray matter and, for example, if the neighboring fibres of the pyramidal tract be affected there may be in addition a spastic paralysis of the muscles whose centres lie in the lower levels of the cord. The degree of such a paralysis depends upon the intensity of the lesion of the pyramidal tract and may vary

Bramwell - 200 cases ave. dur. 12 years Shl. 7 yrs
Cayot 37 yrs.

from a slight weakness in dorsal flexion of the ankle to an absolute paralysis of all the muscles below the lesion. Again, if the afferent tracts of the cord are affected sensory symptoms may be added to the motor palsy. There may be disturbances of pain and temperature sense alone or touch also may be affected. This, however, is more rare except in serious lesions. The upper border of disturbed sensation often indicates most clearly the level of the disease, especially when this is in the thoracic region where the corresponding level of motor paralysis is not easily demonstrated. It is unusual for cutaneous anæsthesia in organic lesions of the cord to extend above the level of the second rib and the tip of the shoulder, for this represents the lower border of the skin-field of the fourth cervical (see sensory charts), and as the chief centre for the diaphragm lies in this segment, a lesion at this level sufficiently serious to cause sensory disturbances would probably occasion motor paralyses as well and would entirely shut off the movements necessary for respiration. It is to be noted that the demonstrable upper border of the anæsthetic field may not quite reach that which represents the level of the lesion. This is due to the functional overlapping of the segmental skin-fields (Sherrington) and applies more to touch than to pain and temperature. There is often a narrow zone of hyperæsthesia above the anæsthetic region.

Complete Transverse Lesions.—When the transverse lesion is total and the lower part of the cord is cut off entirely from above, there is complete sensory and motor paralysis to the segmental level of the injury, and the tendon reflexes, whose centres lie below, are lost instead of being exaggerated, as they are apt to be in case the lesion is a focal one. The symptomatology of total transverse lesions in man has thus been given by Collier. (1) Total flaccid paralysis of muscles below the level of the lesion. (Spastic paralysis indicates that the lesion is incomplete.) (2) Permanent abolition of the knee-jerk and other deep reflexes supplied by the lower segments of the cord. (3) A rapid wasting of the paralyzed muscles with a loss of the faradic excitability. (4) The sphincters lose their tone and there is dribbling of urine. (5) There is total anæsthesia to the level of the lesion (the zone of hyperæsthesia is rarer). (6) The only sign of self-action remaining is in the occasional presence, though in reduced degree, of certain skin reflexes such as the plantar reflex with its dorsal flexor response in the great toe.

Unilateral Lesions.—The motor symptoms, which follow lesions limited to one lateral half of the cross section of the spinal cord, are confined to one side of the body; they are on the same side as the lesion. At the level of the lesion, owing to destruction of cell bodies of the lower system of neurones, there will be found flaccid paralysis and atrophy of those muscles whose centres of innervation happen to lie at this level. Owing to degeneration of the pyramidal tract, the muscles whose centres be at lower levels are also paralyzed, but they retain their normal electrical reactions, become spastic, and do not atrophy to any great degree.

The sensory symptoms are peculiar. On the side of the lesion corresponding to the segment or segments of the cord involved there is a zone of anæsthesia to all forms of sensation. Below this there is no loss in the perception of pain, temperature, or touch. Indeed, hyperæsthesia has been described. Muscle sense is disturbed, and the ability to appreciate the size, consistency, weight, and shape of an object. On the side opposite to the lesion and nearly up to its

level there is complete loss of perception for pain and temperature and there may be some dulling of tactile sense as well.

The following table, slightly modified from Gowers, illustrates the distribution of these symptoms in a complete hemi-lesion of the cord:

Cord

Zone of cutaneous hyperæsthesia. Zone of cutaneous anæsthesia. Lower segment type of paralysis with atrophy.	Lesion.	
Upper segment type of paralysis. Hyperæsthesia of skin. Muscular sense and allied sensations impaired. Reflex action first lessened and then increased. Surface temperature raised.		Muscular power normal. Loss of sensibility of skin to pain and temperature. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

This combination of symptoms was first recognized by Brown-Séquard, after whom it has been named. It is common in syphilitic diseases of the cord, tumors and stab-wounds, and is not infrequently associated with syringomyelia and hæmorrhages into the cord. It is only in exceptional cases, of course, that the lesion is absolutely limited to the hemi-section of the cord and the symptoms consequently may vary somewhat in degree.

II. AFFECTIONS OF THE BLOOD VESSELS

1. CONGESTION

Apart from actual myelitis, we rarely see post mortem evidences of congestion of the spinal cord, and, when we do, it is usually limited either to the gray matter or to a definite portion of the organ. The white matter is rarely found congested, even when inflamed. The gray matter often has a reddish pink tint, but rarely a deep reddish hue, except when myelitis is present. If we know little anatomically of conditions of congestion of the cord, we know less clinically, for there are no features in any way characteristic of it.

2. ANÆMIA

So, too, with this state. There may be extreme grades of anæmia of the cord without symptoms. In chlorosis, for example, there are rarely symptoms pointing to the cord, and there is no reason to suppose that such sensations as heaviness in the limbs and tingling are especially associated with anæmia.

Profound anæmia of the cord follows ligature of the aorta. In experiments made in Welch's laboratory by Herter it was found that within a few moments after the application of the ligature to the aorta paraplegia came on. Paralysis of the sphincters occurred, but less rapidly. Observations

made by Halsted on occlusion of the abdominal aorta in dogs have shown that paraplegia occurs in a large percentage of cases, many of which, however, may recover as the collateral circulation is established. In the fatal cases Gilman found extensive alterations in the cell bodies of the lower part of the cord with degenerations. This condition is of interest in connection with the occasional rapid development of a paraplegia after profuse hæmorrhage, usually from the stomach or uterus. It may come on at once or at the end of a week or ten days, and is probably due to an anatomical change in the nerve elements similar to that produced in Herter's experiments. The degeneration of the dorsal columns of the cord in pernicious anæmia has already been described.

3. EMBOLISM AND THROMBOSIS

Blocking of the spinal arteries by emboli rarely occurs. It may be produced experimentally, and Money found that it was associated with choreiform movements. Thrombosis of the smaller vessels in connection with endarteritis plays an important part in many of the acute and chronic changes in the cord.

4. ENDARTERITIS

It is remarkable how frequently in persons over fifty the arteries of the spinal cord are found sclerotic. The following forms may be met with: (1) A nodular peri-arteritis or endarteritis associated with syphilis and sometimes with gummata of the meninges; (2) an arteritis obliterans, with great thickening of the intima and narrowing of the lumen of the vessels, involving chiefly the medium and larger-sized arteries. Miliary aneurisms or aneurisms of the larger vessels are rarely found in the spinal cord.

5. HÆMORRHAGE INTO THE SPINAL MEMBRANES; HÆMATORRHACHIS

In meningeal apoplexy, as it is called, the blood may lie between the dura mater and the spinal canal—extra-meningeal hæmorrhage—or within the dura mater—intra-meningeal hæmorrhage.

Extra-meningeal hæmorrhage occurs usually as a result of traumatism. The exudation may be extensive without compression of the cord. The blood comes from the large plexuses of veins which may surround the dura. The rupture of an aneurism into the spinal canal may produce extensive and rapidly fatal hæmorrhage.

Intra-meningeal hæmorrhage is a less frequent result of trauma, but in general is perhaps rather more common. It is rarely extensive from causes acting directly on the spinal meninges themselves. Scattered hæmorrhages are not infrequent in the acute infectious fevers, and I have twice, in malignant small-pox, seen much extravasation. Bleeding may occur also in death from convulsive disorders, such as epilepsy, tetanus, and strychnia poisoning, and has been recorded in association with difficult parturition. The most extensive hæmorrhages occur in cases in which the blood comes from rupture of an aneurism at the base of the brain, either of the basilar or vertebral artery. In ventricular apoplexy the blood may pass from the fourth ventricle into the spinal meninges. In cranial fractures, particularly

those of the base of the skull, the resultant hæmorrhage almost always finds its way into the subarachnoid space about the cord and may be demonstrated by the withdrawal of bloody fluid by a lumbar puncture. The procedure is of considerable diagnostic value. On the other hand, hæmorrhage into the spinal meninges may possibly ascend into the brain.

Symptoms.—The symptoms in moderate grades may be slight and indefinite. In the non-traumatic cases the hæmorrhage may either come on suddenly or after a day or two of uneasy sensations along the spine. As a rule, the onset is abrupt, with sharp pain in the back and symptoms of irritation in the course of the nerves. There may be muscular spasms, or paralysis may come on suddenly, either in the legs alone or both in the legs and arms. In some instances the paralysis develops more slowly and is not complete. There is no loss of consciousness, and there are no signs of cerebral disturbance. The clinical picture naturally varies with the site of the hæmorrhage. If in the lumbar region, the legs alone are involved, the reflexes may be abolished, and the action of the bladder and rectum is impaired. If in the thoracic region, there is more or less complete paraplegia, the reflexes are usually retained, and there are signs of disturbance in the thoracic nerves, such as girdle sensations, pains, and sometimes eruption of herpes. In the cervical region the arms as well as the legs may be involved; there may be difficulty in breathing, stiffness of the muscles of the neck, and occasionally pupillary symptoms.

The prognosis depends much upon the cause of the hæmorrhage. Recovery may take place in the traumatic cases, and in those associated with the infectious diseases.

6. HÆMORRHAGE INTO THE SPINAL CORD; HÆMATOMYELIA

Most frequently a result of traumatism, intraspinal hæmorrhage is naturally more common in males, and during the active period of life. Cases have been known to follow cold or exposure; it occurs also in tetanus and other convulsive diseases, and hæmorrhage may be associated with tumors, with syringomyelia or myelitis. A direct injury to the spine, however, from blows or from falls, is by far the most common cause. Thorburn was among the first to point out that acute flexure of the neck, often without attendant fracture or dislocation of the vertebræ, was a form of accident that most commonly preceded these hæmorrhages. The level of the lesion, for this reason, is most frequently in the lower cervical region.

Anatomical Condition.—The extent of the hæmorrhage may vary from a small focal extravasation to one which finds its way in columnar fashion a considerable distance up and down the cord. The bleeding primarily takes place into the gray matter, and this as a rule suffers most, but the surrounding medullated tracts may be thinned out and lacerated. In a case which occurred at the Montreal General Hospital under Wilkins the hæmorrhage occupied a position opposite the region of the fifth and sixth cervical nerves, and on transverse section the cord was occupied by a dark red clot measuring 12 by 5 mm., around which the white substance formed a thin, ragged wall. The clot could be traced upward as far as the second cervical, and downward as far as the fourth thoracic segment.

Virchow's Archiv. f. path. Anat. Band 6. 17.

Thursby: xanthochromia. suggs. of cord tumor in cases of pleur.

2 yellowness. high protein, spec. grav. hyperleucytosis in pneum.
Froriep's syndrome. large endothelioma or sarcoma. roots cerv. & vert. & cauda equina

3. Nonne's syndrome / viscous & globulin [illegible] suggs. of
subarachnoidal cord tumor. & never likely if
has yellow fluid.

Special fluid in empyema of cord. Meyer & Veits, Journ. Dec 5. 11

1. Yellow color. xanthochromia. - in [illegible]

2. Spontaneous coag.

3. Increase in cells. Given only in case of inflammation, emp.
Sometimes necessary part of syndrome

Symps. 1. Meningitis

2. Pachymeningomyelitis of cauda equinalis

3. cord tumors. Some do not show yellow color & coag. in excess
but an increase in fibrin content over that of blood. Tr albs

Meningitis - albumen increased with slight or no lymphocytosis in fluid
emp. of cord - Cases cannot be decided by this and the
Stasis of fluid below site of compression is distinct point [illegible]

Xanthochromia - result from haemoglobin. Coagulation dep.
fluid after it has [illegible]

Symptoms.—As one side of the cord is usually involved more than the other, a type of the Brown-Séquard syndrome is common. The symptoms are sudden in onset, and leave the patient with hyperæsthesia and a paralysis which becomes spastic and is most marked on one side, while anæsthesia, chiefly to pain and temperature, is most marked on the opposite side of the body. Often a most distressing hyperæsthesia, usually a "pins and needles" sensation, may be present for many days, but there is rarely any acute pain of the radiating or root type. As hæmatomyelia is most frequent in the lower cervical region, in addition to the symptoms just mentioned a brachial type of palsy is commonly seen, with flaccid and atrophic paralysis of the muscles innervated from the lowest cervical and first thoracic segments. The hæmorrhage may occur in segments farther down the cord, the lumbar enlargement being affected next in frequency to the lower cervical. The segmental level of the paralysis necessarily would vary accordingly.

The condition may prove rapidly fatal, particularly if the extravasation is bilateral and extends high enough in the cord to involve the centres for the diaphragm. More frequently there is a more or less complete recovery with a residual palsy of the upper extremity and a partial anæsthesia, corresponding to the level of the lesion, and some spasticity of the leg.

Diagnosis.—The diagnosis of the traumatic cases is comparatively easy, and it is important to recognize them, as they are often needlessly subjected to operation under the belief that they are instances of acute compression. The residual symptoms in old cases may closely simulate those seen in syringomyelia.

Treatment.—Absolute rest is important and the patient should be disturbed as little as possible. Special care must be given the skin to prevent bed-sores and to the bladder to prevent cystitis. Treatment of the paralyzed parts should not be begun for six weeks after the hæmorrhage, when electricity, gentle massage, and passive movements are indicated.

III. COMPRESSION OF THE SPINAL CORD

(*Compression Myelitis*)

Definition.—Interruption of the functions of the cord by slow compression.

Etiology.—Caries of the spine, new growths, aneurism, and parasites are the important causes of slow compression. Caries, or Pott's disease, as it is usually called, after the surgeon who first described it, is in the great majority of instances a tuberculous affection. Pressure paralysis from this cause is often associated with angular curvature, but in a large proportion of all the cases the involvement of the cord is due to pachymeningitis externa, to abscess, or in rare cases to direct spicules of bone. There may be a tuberculous pachymeningitis without caries. The paraplegia in Pott's disease may occur without any spinal deformity. These are very difficult cases to recognize, and they are usually associated with pressure of tuberculous material inside the dura. In a few rare cases the paraplegia may be due to a secondary myelitis. In a few cases it is due to syphilis and occasionally to extension of disease from the pharynx. It is most common in early life, but may occur after middle age.

It may follow trauma. Compression occasionally results from aneurism of the thoracic aorta or the abdominal aorta, in the neighborhood of the cœliac axis. Malignant growths frequently cause a compression paraplegia. A retroperitoneal sarcoma or the lymphadenomatous growths of Hodgkin's disease may invade the vertebræ. More commonly, however, the involvement is secondary to scirrhus of the breast. Of parasites, the echinococcus and the cysticercus occasionally occur in the spinal canal.

Symptoms.—These may be considered as they affect the bones, the nerves, and the cord.

VERTEBRAL.—In malignant diseases and in aneurism erosion of the bodies may take place without producing any deformity of the spine. Fatal hæmorrhage may follow erosion of the vertebral artery. In caries, on the other hand, it is the rule to find more or less deformity, amounting often to angular curvature. The compression of the cord, however, is rarely if ever the direct result of this bony kyphosis but is due to the thickening of the dura and the presence of caseous and inflammatory products between this membrane and the bodies of the diseased vertebræ. The spinous processes of the affected vertebræ are tender on pressure, and pain follows jarring movements or twisting of the spine. There may be extensive tuberculous disease without much deformity, particularly in the cervical region. In the case of aneurism or tumor pain is a constant and agonizing feature.

NERVE-ROOT SYMPTOMS.—These result from compression of the nerve roots as they pass out between the vertebræ. In caries, even when the disease is extensive and the deformity great, radiating pains from compression involvement of the roots are rare. Pains are more common in cancer of the spine secondary to that of the breast, and in such cases may be agonizing. There may be acutely painful areas—the *anæsthesia dolorosa*—in regions of the skin which are anæsthetic to tactile and painful impressions. Trophic disturbances may occur, particularly herpes. Pressure on the ventral roots may give rise to wasting of the muscles supplied by the affected nerves. This is most noticeable in disease of the cervical or lumbar regions.

CORD SYMPTOMS.—(*a*) *Cervical Region.*—Not infrequently the caries is high up between the axis and the atlas or between the latter and the occipital bone. In such instances a retropharyngeal abscess may be present, giving rise to difficulty in swallowing. There may be spasm of the cervical muscles, the head may be fixed, and movements may either be impossible or cause great pain. In a case of this kind in the Montreal General Hospital movement was liable to be followed by transient, instantaneous paralysis of all four extremities, owing to compression of the cord. In one of these attacks the patient died.

In the lower cervical region there may be signs of interference with the cilio-spinal centre and dilatation of the pupils. Occasionally there is flushing of the face and ear of one side or unilateral sweating. Deformity is not so common, but healing may take place with the production of a callus of enormous breadth, with complete rigidity of the neck.

(*b*) *Thoracic Region.*—The deformity is here more marked and pressure symptoms are more common. The time of onset of the paralysis varies very much. It may be an early symptom, even before the curvature is manifest, and it is noteworthy that Pott first described the disease that

bears his name as "a palsy of the lower limbs which is frequently found to accompany a curvature of the spine." More commonly the paralysis is late, occurring many months after the curvature has developed. The paraplegia is slow in its development; the patient at first feels weak in the legs or has disturbance of sensation, numbness, tingling, pins and needles. The girdle sensation may be marked, or severe pains in the course of the intercostal nerves. Motion is, as a rule, more quickly lost than sensation. The paraplegia is usually of the spastic type, with exaggeration of the reflexes. Bastian's symptom—abolition of the reflexes—is rarely met with in compression from caries as the transverse nature of the lesion is rarely complete. The paraplegia may persist for months, or even for more than a year, and recovery still be possible.

(*c*) *Lumbar Region.*—In the lower dorsal and lumbar regions the symptoms are practically the same, but the sphincter centres are involved and the reflexes are not exaggerated.

Diagnosis.—The X-ray picture is of first importance. Caries is by far the most frequent cause of slow compression of the cord, and when there are external signs the recognition is easy. There are cases in which the exudation in the spinal canal between the dura and the bone leads to compression before there are any signs of caries, and if the root symptoms are absent it may be extremely difficult to arrive at a diagnosis. Janeway has called attention to persistent lumbago as a symptom of importance in masked Pott's disease, particularly after injury. Brown-Séquard's paralysis is more common in tumor and in injuries than in caries. Pressure on the nerve roots, too, is less frequent in caries than in malignant disease. The cervical form of pachymeningitis also produces a pressure paralysis. Pressure from secondary carcinoma is naturally suggested when spinal symptoms follow within a few years after an operation for cancer of the breast. In paraplegia following tumor of the vertebra secondary to cancer of the breast, and in the erosion of the spine by retroperitoneal growths, the suffering is most intense. The condition has been well termed *paraplegia dolorosa*. I have seen two cases in which the breast tumor had not been recognized.

Treatment.—In compression by aneurism or metastatic tumors the condition is hopeless. In the former the pains are often not very severe, but in the latter morphia is always necessary. On the other hand, compression by caries is often successfully relieved even after the paralysis has persisted for a long period. When caries is recognized early, rest and support to the spine by the various methods now used by surgeons may do much to prevent the onset of paraplegia. When paralysis has occurred, rest with extension gives the best hope of recovery. It is to be remembered that restoration may occur after compression of the cord has lasted for many months, or even more than a year. Cases have been cured by recumbency alone, enforced for weeks or months; the extradural and inflammatory products are absorbed and the caries heals. In earlier days brilliant results were obtained in these cases by suspension, a method introduced by J. K. Mitchell in 1826, and pursued with remarkable success by his son, Weir Mitchell. In recent years the suspension methods in the erect posture have been largely superseded by those of hyperextension during recumbency with the application of plaster jackets to hold the body and spine immovable in the

improved position. Forcible correction of the deformity under anæsthesia as sometimes advocated is not to be recommended; but the gentler partial corrections, perhaps repeated several times with a few weeks' interval, often lead to a rapid disappearance of paralyses through the lessening of the angular deformity of the vertebra. In protracted cases, after these methods have been given a fair trial, laminectomy may become advisable, and has in many instances been successful in relieving paralyses when bloodless methods have failed.

The general treatment of caries is that of tuberculosis—fresh air, good food, ~~cod-liver oil~~, and arsenic.

IV. LESIONS OF THE CAUDA EQUINA AND CONUS MEDULLARIS

The spinal cord extends ~~only~~ to the second lumbar vertebra. Injury, tumors, and caries at or below this level involve not the cord itself, but the bundle of nerves known as the cauda equina and the terminal portion of the cord, the conus medullaris. Much attention has been given to lesions of this part. Fractures and dislocations are common in the lumbo-sacral region, tumors not infrequently involve the filaments of the cauda equina, and some of the nerves are often entangled in the cicatrix of a spina bifida.

A lesion limited to the conus medullaris is rare. A myelitis or a focal hæmatomyelia may be limited to this site with symptoms referable to a lesion of the lowest sacral segments—anæsthesia over the buttocks, perineum, and genitalia, paralysis of the levator ani and the vesical and anal sphincters. Such a focalized lesion has been known to follow a lumbar puncture made between the first and second lumbar vertebræ.

In a fracture or dislocation of the first lumbar vertebra the conus medullaris may be compressed together with the lowest sacral nerves given off from it. It is rare, however, in traumatic cases for the tip of the cord to suffer injury alone without simultaneous involvement of the nerve roots comprising the cauda equina from the second lumbar down. In fracture or dislocation of the fifth lumbar vertebra the sacral roots may alone be involved. Thus in a case which I have reported the patient fell from a bridge and had paralysis of the legs and of the bladder and rectum. When seen sixteen years after the injury, there was slight weakness, with wasting of the left leg; there was complete loss of the function in the ano-vesical and genital centres with anæsthesia in a strip at the back part of the thigh (in the distribution of the small sciatic), and of the perineum, scrotum, and penis. The urethra was also insensitive.

It is sometimes very difficult to differentiate between a lesion, possibly at the first lumbar vertebra, involving the lower part of the spinal cord and one in the sacral region which compromises those peripheral nerves of the cauda equina that are given off from the same segment. This is particularly so in the case of tumors, for in fractures or caries there may be some palpable indication of the seat of trouble. In cauda equina lesions, however, pressure upon the nerve roots is supposed to affect motion much more markedly than sensation, and this discrepancy may be helpful since in the cord lesions

Kennedy Hastings. Jan 13. 11 - Dis. of nerves of lower extremities

pain - from ankle. sharp. shooting & burning - in backs of thighs & calves
more called early. later in both legs - hot feet legs always worst
tingling & numbness - atrophy of ant. tibial muscles then of dorsi-
flexion of ankle - spasticity of legs - more on rgt. side

Sensory disturb - objective - more on lower roots. sacral always & most
less - roots less -

more lower sacral involved - more complete objective disturb -
bladder & rectum no disorder - few cases had such changes Babinski -
abdom. reflexes present in all - Kernig's both sides no clonus

themselves the motor and sensory disturbances are more apt to have a correspondingly segmental distribution.

The table and figures given in the general introduction will be found useful in determining the nerve fibres and segments involved in these cases of injury of the cauda equina.

V. TUMORS OF THE SPINAL CORD AND ITS MEMBRANES

Morbid Anatomy.—New growths may grow in the cord or in its membranes, or may extend into them from the spine. These invading growths are the more common and have been touched upon in a previous section. Here the primary spinal growths only will be considered.

Schlesinger's tabulation of 400 cases shows that meningeal tumors are considerably more common than medullary or true cord tumors. Solitary tubercles are by far the most frequent medullary growths. The meningeal tumors may be either intra- or extradural and the intradural sarcomata or fibromata—it is often difficult to tell under which of these terms they should be classified—are by far the most common. This is important because these particular growths remain for a long time non-infiltrating and offer most favorable opportunities for surgical treatment. In the extradural space echinococcus cysts are in some countries frequently found. They are usually multiple, and, indeed, most of the other forms of tumor may be multiple. A lipoma, psammoma, myxoma, neuroma, and other varieties of growth may be met with. Gummata and gliosarcomata are not infrequent and usually involve both the cord and the meninges.

Tumors are more commonly situated on the lateral and dorsal surfaces of the cord, but there is no level of the spine in which they may not occur.

The effects of tumor on the functions of the cord are varied. Slow compression is usually produced by growths external to the cord, and it is remarkable what a high grade of compression the cord will bear without serious interference with its functions. In cases of prolonged interruption of function ascending and descending degenerations occur. Tumors developing within the cord may lead to syringomyelia.

Symptoms.—These will naturally vary a good deal with the segment involved and with the degree of pressure and the extent of implication of the nerve roots. Neuralgic pains which persist over a particular territory, and a slowly progressive paralysis which may at first suggest a Brown-Séquard syndrome, should always make one suspect a spinal growth.

The symptoms of the commoner intradural tumors are as follows: Radiating (root) pains from the level of the lesion; segmental atrophy from pressure on the ventral horns; weakness of the leg, going on to paralysis, at first only on the side occupied by the growth, and due to pyramidal tract involvement; sensory disturbances on the opposite side, first affecting pain and temperature sense; with increase of symptoms the crossed type of paralysis is lost and motor palsy occurs on both sides with great increase of reflexes; even in advanced cases the sensory paralysis rarely becomes quite complete, since some tactual transmission from the lower sacral segments usually persists; spasmodic, painful, jerking movements of the lower extremities are very characteristic of the

advanced cases. These symptoms will vary naturally with the character of the growth, its segmental level, place of origin, and other factors, but in no other disease is there the same coincidence of a gradual compression paraplegia and persistent radiating pain. In some cases pain may be elicited by deep pressure alongside the spinous processes at the level of the growth, and the patient, by sudden exertion, or by straining, coughing, or sneezing, may greatly increase it.

Diagnosis.—When constant and severe root pains are associated with a progressive paralysis, the diagnosis may be easily made. Caries may cause identical symptoms, but the radiating pains are rarely so severe. Cervical meningitis simulates tumor very closely, and in reality produces identical effects, but the very slow progress and the bilateral character from the outset may be sufficient to distinguish it. In chronic transverse myelitis the symptoms, according to Gowers, may resemble tumor very closely and present radiating pains, a sense of constriction, and progressive paralysis. Syringomyelia, too, may give a similar picture. A radiogram may be of diagnostic aid in case the vertebræ are infiltrated by the growth.

The nature of the tumor can rarely be indicated with precision. With a marked syphilitic history gumma may naturally be suspected, or, with coexisting tuberculous disease, a solitary tubercle.

Treatment.—If the possibility of syphilitic infection is present the iodide of potassium should be given in large and increasing doses. For the severe pains counter-irritation is sometimes beneficial, particularly the thermocautery; morphia is, however, often necessary. A successful laminectomy offers the only hope of relief in case the lesion prove to be non-syphilitic. Since Horsley's first brilliant operation there have been scores of cases of successful extirpation of spinal cord tumors. The intradural fibrosarcomata are the most favorable cases and complete restoration of function in the cord may follow the removal of the tumor. In the infiltrating growths the nerve roots may be divided, or, as has been suggested, even the cord itself sectioned for the relief of the agonizing pain, but ultimate cure is hopeless in malignant growths of this character.

VI. SYRINGOMYELIA

Definition.—A gliomatous new growth about the central canal of the spinal cord, with cavity formation.

Etiology and Morbid Anatomy.—Syringomyelia must be distinguished from dilatation of the central canal—hydromyelus—slight grades of which are not very uncommon either as a congenital condition or as a result of the pressure of tumors. The cavity of syringomyelia has a variable extent in the cord, sometimes running the entire length, but in many cases involving only the cervical and thoracic regions or a more limited area. It is usually in the dorsal portion of the cord and may extend only into one dorsal cornu. The transverse section may be oval or circular or narrow and fissure like. It varies at different levels. The condition is now regarded as a *gliosis*, a development of embryonal neurogliar tissue in which hæmorrhage or degeneration takes place with the formation of cavities.

Of 190 cases, 133 were in men, 57 in women (Schlesinger). A large majority of the cases begin before the thirtieth year. The disease has been met with in three members of the same family.

Symptoms.—The clinical features are extremely complex. In the classical form there are irregular pains, chiefly in the cervical region; muscular atrophy comes on, which may be confined to the arms, or sometimes extends to the legs. The reflexes are increased and a spastic condition develops in the legs. Ultimately the clinical picture may be that of an amyotrophic lateral sclerosis. The tactile sensation is usually normal and the muscular sense is retained, but painful and thermic sensations are not recognized, or there may be in rare instances complete anæsthesia of the skin and of the mucous membranes. This combination of loss of painful and thermic sensations with paralysis of an amyotrophic type is characteristic, but not pathognomonic of the disease. The special senses are usually intact and the sphincters uninvolved. Trophic troubles are not uncommon. Owing to the loss of the pain and heat sensations, the patients are apt to injure themselves. Scoliosis also may be present. The loss of painful and thermic impressions is due to the fact that these pass to the brain in the peri-ependymal gray matter, particularly that portion in the dorsal roots, which is almost constantly involved in syringomyelia. The tactile sensation is retained because the postero-lateral columns are uninvolved.

Schlesinger, in his monograph, recognizes the following types: (1) With the classical features above described, which may begin in the cervical or lumbar regions; (2) a motor type, with the picture of an amyotrophic or a spastic paralysis—the sensation may be undisturbed for years; (3) with predominant sensory features, simulating hysterical hemiplegia, or with general pain and temperature anæsthesia; (4) with pronounced trophic disturbances—to this type belong the cases described as Morvan's disease, an affection characterized by neuralgic pains, cutaneous anæsthesia, and painless, destructive whitlows; and (5) the tabetic type, either a combination of the symptoms of tabes in the lower, and of syringomyelia in the upper extremities, or a pure tabetic symptom-complex, due to invasion of the dorsal columns by the gliosis. Arthropathies occur in about 10 per cent. of the cases.

Diagnosis.—In typical cases the diagnosis is easy. The combination of an amyotrophic paralysis, the picture of progressive muscular atrophy of the Aran-Duchenne type, with retention of tactile and loss of thermic and painful sensation, is probably pathognomonic of the disease. Of affections with which it may be confounded, anæsthetic leprosy is the most important, since the anæsthesia and the wasting may closely simulate it; but, as a rule, in leprosy trophic changes are more or less marked. There is often loss of phalanges and there is no characteristic dissociation of sensory impressions.

Treatment.—This is unsatisfactory, but in a few cases the X-rays have given great relief, particularly to the stiffness.

VII. ACUTE MYELITIS

Etiology.—Acute myelitis results from many causes, and may affect the cord in a limited or extended portion—the gray matter chiefly, or the gray and white matter together. It is met with: (*a*) As an independent affection

following exposure to cold, or exertion, and leading to rapid loss of power with the symptoms of an acute ascending paralysis. (*b*) As a sequel of the infectious diseases, such as small-pox, typhus, measles, and gonorrhœa. (*c*) As a result of traumatism, either fracture of the spine or very severe muscular effort. Concussion without fracture may produce it, but this is rare. Acute myelitis, for instance, scarcely ever follows railway accidents. (*d*) In diseases of the bones of the spine, either caries or cancer. This is a more common cause of localized acute transverse myelitis than of the diffuse affection. (*e*) In disease of the cord itself, such as tumors and syphilis; in the latter, either in association with gummata, in which case it is usually a late manifestation; or it may follow within a year or eighteen months of the primary affection.

Morbid Anatomy.—In localized acute myelitis affecting white and gray matter, as met with after accident or an acute compression, the cord is swollen, the pia injected, the consistence greatly reduced, and on incising the membrane an almost diffluent material may escape. In less intense grades, on section at the affected area, the distinction between the gray and white matter is lost, or is extremely indistinct. The chief features have already been described in discussing the epidemic form.

Histologically the nerve fibres are much swollen and irregularly distorted, the axis-cylinders are beaded, the myelin droplets are abundant, and the laminated bodies known as corpora amylacea may be seen. Changes in the blood vessels are striking: the smaller veins are distended and may show varicosities. The perivascular lymph spaces contain numerous leucocytes, and the smaller arteries themselves are frequently the seat of hyaline thrombi. The ganglion cells are swollen and irregular in outline, the protoplasm is extremely granular and vacuolated, and the nuclei, though usually invisible, may show signs of division, and the processes of the cells are not seen. The acute, inflammatory, hyperæmic or red softening is succeeded by stages in which the affected area becomes more yellow from gradual alteration of the blood pigment, and finally white in color from the advancing fatty degeneration.—In cases of compression myelitis, a sclerosis may gradually be produced with the anatomical picture of a chronic diffuse myelitis.

Symptoms.—(*a*) Acute Diffuse Myelitis.—This form is in the epidemic poliomyelitis, or occurs in connection with syphilis or one of the infectious diseases, or is seen in a typical manner in the extension from injuries or from tumor. The onset, though scarcely so abrupt as in hæmorrhage, may be sudden; a person may be attacked on the street and have difficulty in getting home. In some instances, the onset is preceded by pains in the legs or back, or a girdle sensation is present. It may be marked by chills, occasionally by convulsions; fever is usually present from the beginning—at first sight, but subsequently it may become high.

The *motor* functions are rapidly lost, sometimes as quickly as in Landry's ascending paralysis. The paraplegia may be complete, and, if the myelitis extends to the cervical region, there may be impairment of motion, and ultimately complete loss of power in the upper extremities as well. The sensation is lost, but there may at first be hyperæsthesia. The reflexes in the initial stage are increased, but in acute central myelitis, unless limited in extent to the thoracic and cervical regions, the reflexes are usually abolished. The rectum and bladder are paralyzed. Trophic disturbances are marked; the mus-

cles waste rapidly; the skin is often congested, and there may be localized sweating. The temperature of the affected limbs may be lowered. Acute bed-sores may occur over the sacrum or on the heels, and sometimes a multiple arthritis is present. In these acute cases the general symptoms become greatly aggravated, the pulse is rapid, the tongue becomes dry; there is delirium, the fever increases, and may reach 107° or 108° F.

The course of the disease is variable. In very acute cases death follows in from five to ten days. The cases following the infectious diseases, particularly the fevers and sometimes syphilis, may run a milder course.

The *diagnosis* of this variety of acute myelitis is rarely difficult. In common with the acute ascending paralysis of Landry, and with certain cases of multiple neuritis, it presents a rapid and progressive motor paralysis. From the former it is distinguished by the more marked involvement of sensation, the trophic disturbances, the paralysis of bladder and rectum, the rapid wasting, the electrical changes, and the fever. From acute cases of multiple neuritis it may be more difficult to distinguish, as the sensory features in these cases may be marked, though there is rarely, if ever, in multiple neuritis complete anæsthesia; the wasting, moreover, is more rapid in myelitis. The bladder and rectum are rarely involved—though in exceptional cases they may be —and, most important of all, the trophic changes, the development of bullæ, bed-sores, etc., are not seen in multiple neuritis.

(*b*) Acute Transverse Myelitis.—The symptoms naturally differ with the situation of the lesion.

(1) Acute transverse myelitis in the *thoracic region,* the most common situation, produces a very characteristic picture. The symptoms of onset are variable. There may be initial pains or numbness and tingling in the legs. The paralysis may set in quickly and become complete within a few days; but more commonly it is preceded for a day or two by sensations of pain, heaviness, and dragging in the legs. The paralysis of the lower limbs is usually complete, and if at the level, say, of the sixth thoracic vertebra, the abdominal muscles are involved. Sensation may be partially or completely lost. At the onset there may be numbness, tingling, or even hyperæsthesia in the legs. At the level of the lesion there is often a zone of hyperæsthesia, which is discovered by passing a test tube containing hot water along the spine, when the sensation of warmth changes to one of actual pain. A girdle sensation may occur early, and when the lesion is in this situation it is usually felt between the ensiform and umbilical regions. The reflex functions are variable. There may at first be abolition of the reflexes; subsequently, those which pass through the segments lower than the one affected may be exaggerated and the legs may take on a condition of spastic rigidity. It does not always happen, however, that the reflexes are increased here, for in a total transverse lesion of the cord they are usually entirely lost, as first pointed out by Bastian. That this is not due to the preliminary shock is shown by the fact that the abolition of the reflexes may be permanent. The muscles become extremely flabby, waste, and lose their faradic excitability, and the sphincters lose their tone. The temperature of the paralyzed limbs is variable. It may at first rise, then fall and become subnormal. Lesions of the skin are not uncommon, and bed-sores are apt to form. There is at first retention of urine and subsequently spastic incontinence. If the lumbar centres are involved, there are

from the outset vesical symptoms. The urine is alkaline in reaction and may rapidly become ammoniacal. The bowels are constipated and there is usually incontinence of the fæces. Some writers attribute the cystitis associated with transverse myelitis to disturbed trophic influence.

The course of complete transverse myelitis depends a good deal upon its cause. Death may result from extension. Segments of the cord may be completely and permanently destroyed, in which case there is persistent paraplegia. The pyramidal fibres below the lesion undergo the secondary degeneration, and there is an ascending degeneration of the dorsal median columns. If the lower segments of the cord are involved the legs may remain flaccid. In some instances a transverse myelitis of the thoracic region involves the ventral horns above and below the lesion, producing flaccidity of the muscles, with wasting, fibrillar contractions, and the reaction of degeneration. More commonly, however, in the cases which last many months there is more or less rigidity of the muscles with spasm or persistent contraction of the flexors of the knee.

(2) *Transverse Myelitis of the Cervical Region.*—If the lesion is at the level of the sixth or seventh cervical nerves, there is paralysis of the upper extremities, more or less complete, sometimes sparing the muscles of the shoulder. Gradually there is loss of sensation. The paralysis is usually complete below the point of lesion, but there are rare instances in which the arms only are affected, the so-called cervical paraplegia. In addition to the symptoms already mentioned there are several which are more characteristic of transverse myelitis in the cervical region, such as the occurrence of vomiting, hiccough, and slow pulse, which may sink to 20 or 30, pupillary changes—myosis—sometimes attacks of dysphagia, dyspnœa, or syncope.

Treatment of Acute Myelitis.—In the rapidly advancing form due either to a diffuse inflammation in the gray matter or to transverse myelitis, the important measures are scrupulous cleanliness, care and watchfulness in guarding against bed-sores, the avoidance of cystitis, either by systematic catheterization or, if there is incontinence, by a carefully adjusted bed urinal, or the use of antiseptic cotton-wool repeatedly changed. In an acute onset in a healthy subject the spine may be cupped. Counter-irritation is of doubtful advantage. Chapman's ice-bag is sometimes useful. No drugs have the slightest influence upon an acute myelitis, except in subjects with well-marked syphilis, in which case mercury and potassium iodide should be given energetically. Tonic remedies, such as quinine, arsenic, and strychnia, may be used in the later stages. When the muscles have wasted, massage is beneficial in maintaining their nutrition. The patient should make every effort to perform muscular movements himself and thus aid improvement. Electricity should not be used in the early stages of myelitis. It is of no value in the transverse myelitis in the thoracic region with retention of the nutrition in the muscles of the leg.

E. DIFFUSE AND FOCAL DISEASES OF THE BRAIN

I. TOPICAL DIAGNOSIS

Only certain regions of the brain give localizing symptoms. These are the cortical motor centres and the associated sensory centres, the speech centres,

the centres for the special senses, and the tracts which connect these cortical areas with each other and with other parts of the nervous system.

The following is a brief summary of the effects of lesions from the cortex to the spinal cord:

The Cerebral Cortex.—(*a*) *Destructive lesions* of the motor cortex cause *paralysis* in the muscles of the opposite side of the body. The paralysis is at first flaccid, but the spastic condition subsequently develops. The extent of the paralysis depends upon that of the lesion. It is apt to be limited to the muscles of the head or of an extremity, giving rise to the cerebral monoplegias. One group of muscles may be much more affected than others, especially in lesions of the highly differentiated area for the upper extremity. It is uncommon to find all the muscle groups of an extremity equally involved in cortical monoplegia. In small bilateral symmetrical lesions monoplegia of the tongue may result without paralysis of the face. A lesion may involve centres lying close together or overlapping one another, thus producing associated monoplegias—e. g., paralysis of the face and arm, or of the arm and leg, but not of the face and leg without involvement of the arm. Very rarely the whole motor cortex is involved, causing paralysis of the opposite side—cortical hemiplegia. Usually in such instances there is marked recovery, so that only a monoplegia persists.

Adjoining and posterior to the motor area is believed to be the region of the cortex in which the impulses concerned in general bodily sensation (cutaneous sensibility, muscle sense, visceral sensations) first arrive (the somæsthetic area). Combined with the muscular weakness there is usually some disturbance of sensations, particularly of those of the muscular sense. In lesions of the superior parietal lobe the stereognostic sense is very often affected. For example, when a coin or a knife is placed in the hand of the affected limb, the patient's eyes being closed, it is not recognized, owing to inappreciation of the form and consistence of the object, and this even though the slightest tactile stimulus applied to the fingers or surface of the hand is felt and may be correctly localized. The sense of touch, pain, and temperature may be lowered, but usually not markedly unless the superior and inferior parietal lobules are involved in subcortical lesions. Paræsthesias and vaso-motor disturbances are common accompaniments of paralyses of cortical origin.

(*b*) *Irritative lesions* cause localized spasms. The most varied muscle groups corresponding to particular movement forms may be picked out. If the irritation be sudden and severe, typical attacks of Jacksonian epilepsy may occur. These convulsions are often preceded and accompanied by subjective sensory impressions. Tingling or pain, or a sense of motion in the part, is often the *signal symptom* (Seguin), and is of great importance in determining the seat of the lesion.

When lesions are often both destructive and irritative, there are combinations of the symptoms produced by each. For instance, certain muscles may be paralyzed, and those represented near them in the cortex may be the seat of localized convulsions, or the paralyzed limb itself may be at times subject to convulsive spasms, or muscles which have been convulsed may become paralyzed. The close observation of the sequence of the symptoms in such cases often makes it possible to trace the progress of a lesion involving the motor cortex. In these cases the most frequent cause is a developing tumor, though

sometimes local thickenings of the membranes of the brain, small abscesses, minute hæmorrhages, or fragments of a fractured skull must be held responsible.

In another section lesions involving the centres for the special senses are considered, and we shall simply refer to them here. The symptoms caused by lesions of the speech centres will be described under aphasia, and it is only necessary to note here the near situation of the motor speech area (Broca's centre) in the left inferior frontal convolution to the centres for the face and tongue on that side, and the nearness of the supposed centre for writing to that of the hand and arm, and to state that motor aphasia is often associated with paralysis of the right side of the face and the right arm. Accompanying the paralysis, following a Jacksonian fit, of the right face or arm there is often a transient motor aphasia.

According to Flechsig, the sensori-motor centres are limited to tolerably circumscribed areas in the cortex, which differ from other portions in that they are provided with projection fibres which connect them with lower centres. The remaining areas of the cortex, amounting, he believes, to about two-thirds of the whole, are devoid of projection fibres and are concerned entirely in associative activities. These latter areas, the "association centres" of Flechsig, are three in number: (1) The anterior association centre, including the whole of the frontal lobe in front of the somæsthetic area; (2) the middle association centre, corresponding to the cortex of the island of Reil; and (3) the large, posterior association centre, including the præcuneus, the superior and inferior parietal lobules, the supramarginal and angular gyri, and the whole of the temporal and occipital lobes except the auditory and visual sensory areas.

Flechsig attributes the higher psychic functions, especially those connected with the personality of the individual, to the anterior association centres, while the intellectual activities which have to do with knowledge of the external world he believes correspond to the functions of the large posterior association centre. Whether these views be true, and, if so, in how far they may be applied practically in the localization of diseases, especially of the mind, the future has to decide.

Centrum Semiovale.—Lesions in this part may involve either projection fibres (motor or sensory) or association fibres. If involvement of the motor path cause paralysis, this has the distribution of a cortical palsy when the lesion is near the cortex, and of a paralysis due to a lesion of the internal capsule when it is near that region. These lesions of the motor fibres may be associated with symptoms due to interruption in the other systems of fibres running in the centrum semiovale; there may be sensory disturbances—hemianæsthesia and hemianopia—and if the lesion is in the left hemisphere one of the different forms of aphasia may accompany the paralysis.

Corpus Callosum.—This may be congenitally absent without symptoms. An acute lesion involving a large portion of the corpus callosum may, however, yield symptoms suggestive of its localization in this region. In the case recorded by Reinhard, in which the situation of the lesion was suspected ante mortem, there was a disturbance of equilibration (without vertigo) and of the synergetic movements of both halves of the body. The autopsy revealed a gliosarcoma which had destroyed the posterior three-fourths of the corpus

callosum. In Bristowe's 4 cases there existed, as symptoms common to all, pain in the head and partial or complete hemiplegia, with gradual extension of the paralysis to the opposite side of the body. Toward the end of life there were disturbance of speech, difficulty in deglutition, incontinence of urine and faeces, and dementia. Here the symptoms have in them nothing that can be looked upon as pathognomonic; indeed, many of the phenomena were doubtless dependent upon involvement of the projection and association fibres of the centrum semiovale.

In animals in which the corpus callosum has been cut experimentally progressive emaciation has been mentioned as a characteristic phenomenon.

Internal Capsule (Fig. 13).—Through this pass within a rather narrow area all, or nearly all, of the projection fibres (both motor and sensory) which are connected with the cerebral cortex. It is divided into an anterior limb, a knee, and a posterior limb, the latter consisting of a thalamo-lenticular portion (its anterior two-thirds) and a retro-lenticular portion (its posterior third). In considering the effects of a given focal lesion involving the fibres of the internal capsule, it is not to be forgotten that the relations of the two limbs of the capsule to one another and to the knee vary considerably in different horizontal planes. Much of the confusion in the bibliography is dependent upon neglect to describe the horizontal level of the lesion, as well as its situation in an antero-posterior direction. The principal bundle passing through the anterior limb of the capsule is that which connects the frontal gyri and the medial bundle in the base of the peduncle (crus) with the nuclei of the pons. These fibres are centrifugal, and innervate chiefly the lower motor nuclei governing bilaterally innervated muscles, especially those of the eyes, head, neck, and probably those of the mouth, tongue, and larynx. In lower horizontal planes these fibres are situated near the knee of the capsule. It is the region of the knee of the capsule which transmits especially the fibres passing from the cerebral cortex to the nuclei of the facial, hypoglossal, and third nerves. The path which supplies the nuclei governing the muscles used in speech passes through the knee.

The pyramidal tract goes through the thalamo-lenticular portion of the capsule. The motor fibres are arranged according to definite muscle groups, or rather movement forms, those for the movements of the arm being anterior to those for the leg. The number of fibres for a given muscle group corresponds rather to the degree of complexity of the movements than to the size of the muscles concerned. Thus the areas for the fingers and toes are relatively large.

The fibres to the somæsthetic area of the cortex—that is, those from the centro-lateral group of nuclei of the thalamus and the tegmental radiations—carrying impulses concerned in general bodily sensation, pass upward through the posterior part of the thalamo-lenticular portion of the capsule. Some of these fibres pass through the anterior two-thirds of the posterior limb alongside of the fibres of the pyramidal tract.

Through the retro-lenticular portion of the posterior limb, opposite the posterior third of the lateral surface of the thalamus, pass (1) the fibres carrying impulses concerned in the sensations of the opposite visual field (optic radiation from the lateral geniculate body to the visual sense area in the occipital cortex); (2) the fibres carrying impulses concerned in auditory sensations

(radiation from the medial geniculate body to the auditory sense area in the cortex of the temporal lobe); (3) the fibres (probably centrifugal) connecting the cortex of the temporal lobe with the nuclei of the pons.

With this preliminary knowledge concerning the internal capsule, it is not difficult to understand the symptoms which result when it is diseased.

Since here all the fibres of the upper motor segment are gathered together in a compact bundle, a lesion in this region is apt to cause complete hemiplegia of the opposite side, followed later by contractures; and if the lesion involves the hinder portion of the posterior limb there is also hemianæsthesia, including even the special senses (Fig. 13). As a rule, however, lesions of the internal capsule do not involve the whole structure. The disease usually affects ~~mainly either~~ the anterior or posterior portions, and even in instances in which at first the symptoms point to total involvement there is a disappearance often of a large part of the phenomena after a short time. Thus, when the pyramidal tract is destroyed (lesion of the thalamo-lenticular portion of the capsule) the arm may be affected more than the leg, or *vice versa*. The facial paralysis is usually slight, though if the lesion be well forward in the capsule the paralysis of the face and tongue may be marked.

Hemianæsthesia alone without involvement of the motor fibres~~, due to disease of the capsule,~~ is rare. There is usually also at least partial paralysis of the leg. When the retro-lenticular portion of the capsule is destroyed the hemianæsthesia is accompanied by hemianopsia, disturbances of hearing, and sometimes of smell and taste. The occurrence of hemianæsthesia with pain, hémichorea, marked tremor, or hemiathetosis—thalamic syndrome—after a capsular hemiplegia points to the involvement of the thalamus or of the hypothalamic region.

Charcot and others have described cases in which as a result of disease of the internal capsule there has been paralysis of the face and leg without involvement of the arm. In such instances the lesion is linear, extending from the posterior part of the anterior limb of the internal capsule backward and lateralward to the leg region in the posterior limb of the capsule, the region for the arm escaping.

Capsular lesions when pure are not usually accompanied by aphasic symptoms, alexia, or agraphia. A "subcortical" motor aphasia may result if the lesion is bilateral, as in pseudo-bulbar paralysis, or if on the left side it is so extensive as to destroy the fibres connecting Broca's convolution with the opposite hemisphere, as well as the pyramidal fibres on the same side.

Crura (Cerebral Peduncles).—From this level through the pons, medulla, and cord the upper and lower motor segments are represented, the first by the fibres of the pyramidal tracts and by the fibres which go from the ~~cerebral~~ cortex to the nuclei of the cerebral nerves, the latter by the motor nuclei and the nerve fibres arising from them. Lesions often affect both motor segments, and produce paralyses having the characteristics of each. Thus a single lesion may involve the pyramidal tract and cause a spastic paralysis on the opposite side of the body, and also involve the nucleus or the fibres of one of the cerebral nerves, and so produce a lower segment paralysis on the same side as the lesion —crossed paralysis. In the crus the third and fourth cerebral nerves run near the pyramidal tract, and a lesion of this region is apt to involve them or their

nuclei, causing partial paralysis of the muscles of the eye on the same side as the lesions, combined with a hemiplegia of the opposite side (Fig. 10, 3).

The optic tract also crosses the crus and may be involved, giving hemianopsia in the opposite halves of the visual fields.

If the tegmentum be the seat of a lesion which does not involve the base of the peduncle (or pes) there may be disturbances of cutaneous and muscular sensibility, ataxia, disturbances of hearing, or oculo-motor paralysis. An oculo-motor paralysis of one side, accompanied by a hemiataxia of the opposite side, appears to be especially characteristic of a tegmental lesion.

Corpora Quadrigemina.—Anatomical studies point to the view that the superior colliculus (anterior quadrigeminal body) represents the most important subcortical central organ for the control of the eye-muscle nuclei. This is supported to a certain extent by clinical evidence, though as yet but few cases have been carefully studied. Sight is only slightly, if at all, disturbed when the superior colliculus is destroyed. The pupil is usually widened, and the pupillary reaction, both to light and on accommodation, interfered with. Apparently actual paralysis of the eye muscles does not occur unless the nucleus of the third nerve ventral to the aqueduct be also injured.

The inferior colliculus (posterior quadrigeminal body), on the other hand, has been shown by anatomical study to be an important way-station in the auditory conduction-path. A large part of the lateral lemniscus ends in its nucleus, and from it emerge medullated fibres which pass through the brachium quadrigeminum inferius to the medial geniculate body. Thence a large bundle runs through the retro-lenticular portion of the internal capsule to the auditory sense area in the cortex of the temporal lobe.

Weinland has collected 19 cases of tumors of the corpora quadrigemina from the bibliography; in 9 of these auditory disturbances were especially noted. Since the central auditory path of each side receive impulses from both ears, lesion of the colliculus on one side may dull the hearing on both sides, though the opposite ear is usually the more defective. Lesion of the inferior colliculus may be accompanied by disturbance of mastication, owing to paralysis of the descending (mesencephalic) root of the trigeminus. The fourth nerve may also be involved. The ataxia which sometimes accompanies lesions of the corpora quadrigemina is probably to be referred to disturbance in conduction in the medial lemniscus.

Pons and Medulla Oblongata.—Lesions involving the pyramidal tract, together with any one of the motor cerebral nerves of this region, cause crossed paralysis. A lesion in the lower part of the pons is apt to cause a lower-segment paralysis of the face on the same side (destruction of the nucleus of the facial nerve or of its root fibres) and a spastic paralysis of the arm and leg on the opposite side (injury to pyramidal tract) (Fig. 10, 4). The abducens, the motor part of the trigeminus, and the hypoglossus nerves may also be paralyzed in the same manner. When the central fibres to the nucleus of the hypoglossus are involved a peculiar form of anarthria results. If the nucleus itself be diseased, swallowing is interfered with.

When the sensory fibres of the fifth nerve are interrupted, together with the sensory tract (the medial lemniscus or fillet) for the rest of the body, which has already crossed the middle line, there is a crossed sensory paralysis—i. e.,

disturbed sensation in the distribution of the fifth on the side of the lesion, and of all the rest of the body on the opposite side.—

A paralysis of the external rectus muscle of one eye and of the internal rectus of the other eye (conjugate paralysis of the muscles which turn the eyes to one side), in the absence of a "forced position" of the eyeballs, is highly characteristic of certain lesions of the pons. In such cases the internal rectus may still be capable of functioning on convergence, or when the eye to which it belongs is tested independently of that in which the external rectus is paralyzed. This form of paralysis is found, as a rule, only when the lesion lies just in front of the abducens or involves the nucleus itself, or includes, besides the root fibres of the abducens, that portion of the formatio reticularis that lies between them and the fasciculus longitudinalis medialis (von Monakow). The cases of conjugate paralysis just referred to may be complicated by other disturbances of the eye-muscle movements, in which case the interpretation of the symptoms may be rendered difficult. The facial nerve is often involved in these paralyses.

In lesions of the pons the patient often has a tendency to fall toward the side on which the lesion is, probably on account of implication of the middle peduncle of the cerebellum (brachium pontis). Still more frequent is the simple motor hemi-ataxia consequent upon lesion of the medial lemniscus, and perhaps of longitudinal bundles in the formatio reticularis. This is often accompanied by a dissociated sensory disturbance, pain and temperature being affected, while touch remains normal. The muscular sense may also be involved. Only when the lesion is very extensive are there disturbances of hearing (involvement of the lateral lemniscus or corpus trapezoideum).

The symptoms produced by involvement of the different cerebral nerves will be considered in detail in another section.

Cerebellum.—The functions of this part of the brain are still under consideration. Luciani, whose monograph is exhaustive, regards it as "an end organ, directly or indirectly related to certain peripheral sensory organs and in direct efferent relationship with certain ganglia of the cerebro-spinal axis, and indirectly with the motor apparatus in general. It is functionally homogeneous, each part exercising the functions of the whole, but having special relations to the muscles of the corresponding side of the body" (Krauss).

Lesions of the lateral lobes affect the corresponding side of the body, while lesions of the middle lobe (vermis) affect both sides. Partial removal is followed by transient muscular weakness; complete removal by extreme incoördination. Its one important function would appear to be the coördination of the muscular movements.

In monkeys the symptoms differ much at different periods after the operation. During the first five or six days irritation phenomena predominate. According to Luciani, there are asthenia, atony of the muscles, and astasia on the side of the body operated upon. The animal can not stand or walk. All these symptoms may gradually disappear in the course of a few months.

The experiments of Risien Russell do not entirely confirm the observations of Luciani. In the first place, the occurrence of asthenia is not constant, and as to atony, while the patellar tendon reflexes are sometimes absent, they are, as a rule, intact in pure cerebellar lesions. There may be even muscular rigidity instead of atony. Russell's experiments make it seem likely that the

arterial and venous blood is subject to change. The circulation within the skull not only differs from the circulation in other parts in its freedom from the effects of atmospheric pressure, but apparently it is not under local vaso-motor control and is in an organ that can only expand slightly. Although nerve fibres have been demonstrated in the walls of the small arteries of the brain, it has not been proved that they cause dilatation or contraction under influences from the vaso-motor centres; indeed, there is little experimental evidence that speaks for, and much that speaks against, this view.

Under ordinary circumstances, the circulation of the brain follows passively the general bodily conditions. When anything increases the force with which the blood enters the skull—i. e., when blood pressure is raised, either by increase in the heart's action or by general vaso-motor effects—more blood passes through the brain in a given time, and it is, as it were, flooded with blood. This active hyperæmia must occur under many circumstances, but it is doubtful whether it causes any symptoms; in fact, it is difficult to see how it, in itself, can do anything but good.

Although without direct vaso-motor control, the circulation of the brain is regulated by the action of the vaso-motor centre on the splanchnic areas and skin. This centre itself shares with the respiratory and cardiac centres the same circulatory conditions as prevail throughout the brain.

Consciousness depends upon a due blood supply to the brain, particularly to the cortex, and life itself depends upon the circulation in the medullary centres. When the blood circulating about these centres is poor in oxygen—i. e., when there is a lack of arterial blood—the arterioles within the splanchnic and skin areas contract under vaso-motor influences, the blood pressure is raised, and the blood enters the brain with unusual force and supplies the capillaries with arterial blood. The extent to which this regulating mechanism can counteract an obstruction to the circulation through these centres has been well shown experimentally by Harvey Cushing. When the general intracranial pressure was raised to arterial blood pressure, instead of the circulation being blocked and the animal dying from anæmia of the brain, as has been stated, he showed that the vaso-motor centres responded with a sufficient rise of blood pressure to overcome the impediment, and so restore the circulation. With every repeated increase of intracranial pressure, there was an answering rise of blood pressure, until, at the end of the experiment, the brain was acting under an intracranial pressure much above the arterial pressure of the animal at the beginning of the experiment, and this pressure had been correspondingly raised to a startling extent. The interesting clinical deductions which Cushing draws from this experiment will be referred to under cerebral hæmorrhage.

When this regulating mechanism is disturbed, serious results may follow. The ordinary fainting fit is an example: Under the influence of emotion the vaso-motor centre is inhibited, and, in consequence, the abdominal blood vessels become dilated, blood pressure falls, and the heart is no longer able to drive the blood back to itself against the force of gravity; the blood accumulates in the abdominal veins, the heart empties, cerebral circulation fails, and unconsciousness occurs. A similar condition may follow the sudden removal of something that has caused pressure on the abdominal vessels for a considerable time, as the withdrawal of the ascitic fluid. In this case the vaso-motor

control influences have not been called on for some time, and the centre itself has taken part in the general weakened condition of the individual, so that, when a sudden demand is made upon it to compensate for the accustomed external support to the blood vessels, it is entirely unable to respond, and the blood collects in the splanchnic vessels, the patient becomes unconscious and may die, having bled to death into his own veins.

While under ordinary circumstances the vaso-motor mechanism and the tonicity of the muscles of the abdominal walls compensate perfectly for the change from the horizontal to the upright position—i. e., for the effect of gravity upon the column of venous blood from the heart to the feet, in asthenic states, as after severe illness, the compensation may be very imperfect. When such is the case, if the patient stands, or, at times, even if he sits up in bed, his heart beats more rapidly, he becomes giddy and may faint. The change in the pulse rate, with a change in position, is a fair indication of the vaso-motor control, for the heart itself endeavors to make up for this incompetence.

Chloroform and, to a less extent, ether tend to induce vaso-motor paralysis, and this is the reason why position is such an important factor in the safety of patients during anæsthesia. The splanchnic circulation, under these circumstances, may, to a certain extent, be supported by bandaging the legs and abdomen and elevating the foot of the bed. Crile's pneumatic operating suit, in which the patient is encased below the chest in an inflatable suit, by means of which pressure on the peripheral and abdominal vessels may be varied, is an attempt to establish an artificial vaso-constructor system under the control of the operator, which can compensate for the paralyzing effects of the anæsthetic, and obviate the necessity of considering position.

The heart itself may become weak from various causes and so be unable to keep the brain properly supplied with arterial blood. The extreme example of this is paralysis of the heart muscles from failure of the coronary circulation, which is immediately followed by unconsciousness and death. In Stokes-Adams disease the cerebral symptoms, attacks of unconsciousness, convulsions, and apoplectiform seizures are due to cerebral anæmia, caused by the temporary cessation of the ventricular systole. When the chest is forcibly compressed the heart may be unable to fill itself with blood, and so unconsciousness, or even death, may follow from failure of the cerebral circulation.

Respiration is an essential part of circulation; this is true not only in the primary sense, that it is through this function that venous is changed into arterial blood, but also in a more truly mechanical sense. With every inspiration the blood is sucked into the heart from the veins, and the descent of the diaphragm, by increasing the pressure on the abdominal veins, tends to force the blood into the heart. During expiration the entrance of the blood into the heart is impeded by the increase in the intra-thoracic pressure. Respiration has direct, but slight, influence upon the blood pressure within the arteries.

The circulation within the skull is very intimately related to respiration. The blood from the brain sinuses passes through the jugular veins directly into the superior vena cava and the columns of blood appear to be uninterrupted by competent valves, so that every change of pressure in the cava is transmitted directly to the sinuses and veins of the brain. Intracranial pressure has been shown to be equal to venous blood pressure within the sinuses

and to follow every change in this. The brain dilates with each pulse-beat, but relatively much more with each expiration. In expiration intrathoracic pressure is increased, and this causes an increase in the pressure within the cava, the jugular, and the brain sinuses. The blood is, as it were, dammed back, venous congestion occurs, intracranial pressure rises, and the brain receives less arterial blood, and the symptoms of cerebral anæmia may follow. Under ordinary conditions these effects are not so pronounced or protracted as to cause marked symptoms, but at times they may be, as when a crying child holds his breath until he becomes unconscious. Here the difficulty which the heart has in filling itself with blood under increased thoracic pressure is also a factor. When the superior vena cava is alone obstructed, as by pressure from a tumor, there may be not the slightest disturbance of the functions. This depends upon the freedom of the cranio-vertebral venous anastomosis, and other paths which allow the blood to reach the heart through the inferior vena cava. Strong respiratory efforts against an obstruction may change intrathoracic pressure very greatly. In forced expiration with the glottis closed, the normal negative pressure becomes markedly positive and may far exceed the normal pressure on the intrathoracic veins, while if the glottis be closed and a strong inspiratory effort be made the pressure may fall far below atmospheric pressure. Intracranial hæmorrhages not infrequently take place during a strong effort with the breath held as when straining at stool, or when lifting a heavy weight, or during a severe coughing spell, all conditions in which, among other things, the flow of the venous blood from the brain to the heart is impeded, and in consequence of which intracranial circulatory conditions are altered in the direction of a rise of venous and capillary pressure. The importance of preventing, as far as possible, any obstruction to respiration during the course of apoplexy will be referred to in a subsequent paragraph.

The venous outlets from the skull are so large and the anastomoses are so free that they must all be obstructed to cause any marked anæmia of the brain, and for this reason thrombosis or ligature of one of the sinuses is not necessarily followed by any symptoms. If all the veins in the neck are compressed, as by a tight band or strong flexion of the neck, the circulation may be impeded to a considerable extent, and this is of definite importance under pathological conditions.

Any one of the arteries may be tied before entering the skull, with but little danger, owing to the freedom of the anastomosis in the circle of Willis, but, as this is subject to variation, the closure should be made slowly. With this precaution, both carotids may be tied if an interval be allowed between the operations.

Obliteration of an artery beyond the circle of Willis is always followed by a disturbance of function of the part of the brain supplied by that artery, and is considered under Embolism and Thrombosis.

2. HYPERÆMIA AND ANÆMIA

Less and less stress is now laid on active hyperæmia as a cause of symptoms. As Leube suggests, the symptoms usually referred to active hyperæmia in the infectious diseases, or in association with hypertrophy of the heart ac-

companying disease of the kidney, are due to the action of toxic agents rather than to changes in the circulation. On the other hand, venous stasis and anæmia of the brain must be a very potent cause of head symptoms. The uncertainty which exists is largely due to the fact that the condition of the blood vessels as seen within the skull after death may bear no relation to that which held sway during life.

The anatomical condition of the brain in anæmia is very striking. The membranes are pale, only the large veins are full, the small vessels over the gyri are empty, and an unusual amount of cerebro-spinal fluid is present. On section both the gray and white matter look extremely pale and the cut surface is moist. Very few *puncta vasculosa* are seen.

The effects of sudden anæmia of the brain are well illustrated by the ordinary fainting fit, and have been described above.

Symptoms.—When the symptoms are the result of hæmorrhage, there are drowsiness, giddiness, inability to stand; flashes of light, dark spots before the eyes, and noises in the ears; the respiration becomes hurried; the skin is cool and covered with sweat; the pupils are dilated, there may be vomiting, headache, or delirium, and gradually, if the bleeding continues, consciousness is lost and death may occur with convulsions. In the more chronic forms of brain anæmia, such as result from the gradual impoverishment of the blood, as in protracted illness or in starvation, the condition known as irritable weakness results. Mental effort is difficult, the slightest irritation is followed by undue excitement, the patient complains of giddiness and noises in the ears, or there may be hallucinations or delirium. These symptoms are met with in an extreme grade as a result of prolonged starvation, and a very similar condition is seen in certain cases of arterio-sclerosis where the brain is poorly nourished.

An interesting set of symptoms, to which the term *hydrencephaloid* was applied by Marshall Hall, occurs in the debility produced by prolonged diarrhœa in children. The child is in a semi-comatose condition with the eyes open, the pupils contracted, and the fontanelle depressed. In the earlier period there may be convulsions. The coma may gradually deepen, the pupils become dilated, and there may be strabismus and even retraction of the head, symptoms which closely simulate those of basilar meningitis.

3. ŒDEMA OF THE BRAIN

Pathology.—In the pathology of brain lesions œdema formerly played a rôle almost equal in importance to congestion. It occurs under the following conditions: In general atrophy of the convolutions, in which case the œdema is represented by an increase in the cerebro-spinal fluid and in that of the meshes of the pia. In extreme venous dilatation from obstruction, as in mitral stenosis or in tumors, there may be a condition of congestive œdema, in which, in addition to great filling of the blood vessels, the substance of the brain itself is unusually moist. The most acute œdema is a local process found around tumors and abscesses. The symptoms of compression following concussion or contusion, as shown by Cannon, are frequently attributable to cerebral œdema due to change in osmotic pressure. An intense infiltration, local or general, may occur in Bright's disease, and to it, as Traube suggested, certain of the uræmic symptoms may be due.

Anatomical Changes.—The anatomical changes are not unlike those of anæmia. When the œdema follows progressive atrophy, the fluid is chiefly within and beneath the membranes. The brain substance is anæmic and moist, and has a wet, glistening appearance, which is very characteristic. In some instances the œdema is more intense and local, and the brain substance may look infiltrated with fluid. The amount of fluid in the ventricles is usually increased.

Symptoms.—The symptoms are in great part those of lessened blood flow, and are not well defined. As just stated, some of the cerebral features of uræmia may depend upon it. Cases have been reported by Raymond, Tennecon, and Dercum, in which unilateral convulsions or paralysis have occurred in connection with chronic Bright's disease, and in which the condition appeared to be associated with œdema of the brain. The older writers laid great stress upon an apoplexia serosa, which may really have been a general œdema of the brain. Inasmuch as the instances in which œdema of the brain occurs are often those in which there is also intoxication, or anæmia, or both, it is probably impossible to say at the bedside definitely which of these possible factors is responsible for the symptoms in a given case.

4. CEREBRAL HÆMORRHAGE

The bleeding may come from branches of either of the two great groups of cerebral vessels—the *basal*, comprising the circle of Willis and the central arteries passing from it and from the first portion of the cerebral arteries, or the *cortical group*, the anterior, middle, and the posterior cerebral vessels. In a majority of the cases the hæmorrhage is from the central branches, more particularly from those which are given off by the middle cerebral arteries in the anterior perforated spaces, and which supply the corpora striata and internal capsules. One of the largest of these branches which passes to the third division of the lenticular nucleus and to the anterior part of the internal capsule, the lenticulo-striate artery of Duret, is so frequently involved in hæmorrhage that it has been called by Charcot *the artery of cerebral hæmorrhage*. Hæmorrhages from this and from the lenticulo-thalamic artery include more than 60 per cent. of all cerebral hæmorrhages. The bleeding may be into the substance of the brain, to which alone the term cerebral apoplexy is applied, or into the membranes, in which case it is termed meningeal hæmorrhage; both, however, are usually included under the terms intracranial or cerebral hæmorrhage.

Etiology.—The conditions which produce degeneration of the blood vessels play the important part.

Age.—The liability increases with each decade. H. M. Thomas, in his analysis of the United States Census Report for 1907, states that the greatest number of cases occurred in the seventh and eighth decades. Cerebral hæmorrhage may be congenital. One of the only recorded instances is that which I reported in a six-month fetus of a woman dead of typhoid fever. The clot was in the left hemisphere and had broken into the ventricle. Apart from meningeal hæmorrhage, which is common as a result of the accidents of birth, hæmorrhage is rare in children, and we had no instance at the Johns Hopkins Hospital during my term of service. Before the fifth decade hæmorrhage is

rare; then in the fifth and sixth decades cases progressively increase in number.

Sex.—There is a marked preponderance of males.

Race.—In the United States the death rate from apoplexy and paralysis in the Report of 1906 was 88.7 per 100,000 of the population. In England and Wales in 1909 the deaths from apoplexy were 502 per million living. Both apoplexy and paralysis seem to be much more prevalent among the negroes.

Heredity.—Formerly thought to be a very important factor, heredity influences the incidence in rendering members of families in which the blood vessels degenerate early more liable to cerebral hæmorrhage. What was formerly known as the apoplectic habitus, or build, is still spoken of, by which we mean a stout, plethoric person of medium size with a short neck.

Special Factors.—Individuals with progressive renal disease and consecutive arterio-sclerosis and hypertrophy of the heart are particularly liable to cerebral hæmorrhage. The causes of arterio-sclerosis, such as alcohol, immoderate eating, prolonged muscular exertion, syphilis, chronic lead poisoning, and gout, are antecedents in many cases. Endocarditis may lead indirectly to apoplexy by causing embolism and aneurism of the vessels of the brain. Cerebral hæmorrhage occurs occasionally in the specific fevers and in such profound alterations of the blood as are met with in leukæmia.

The actual exciting cause is not always evident. The attacks may be sudden without any preliminary symptoms. In other instances violent exertion, particularly straining efforts or overaction of the heart in emotion, may cause a rupture. Many cases occur during sleep. Some instances follow slight trauma. The records of University College Hospital analyzed by Ernest Jones indicate that in none of 123 cases did the attack come on through excessive bodily effort.

Morbid Anatomy.—Direct Changes.—The lesions causing apoplexy are almost invariably in the cerebral arteries, in which the following changes may lead directly to it:

(*a*) The production of miliary aneurisms, rupture of which is the most common cause of cerebral hæmorrhage. The origin of the miliary aneurisms is disputed. Charcot thought they resulted from changes in the *adventitia* (periarteritis). Others find the primary change in the *intima*. The weight of opinion at present, however, is on the side of the view that the *media* is first degenerated. They occur most frequently on the central arteries, but also on the smaller branches of the cortical vessels. On section of the brain substance they may be seen as localized, small dark bodies, about the size of a pin's head. Sometimes they are seen in numbers upon the arteries when carefully withdrawn from the anterior perforated spaces. According to Charchot and Bouchard, who have described them, they are most frequent in the central ganglia. In apoplexy after the fortieth year if sought for they are rarely missed. The actual miliary aneurism, which by its rupture has occasioned the hæmorrhage, may be difficult to find, but if one pours water carefully on the area of hæmorrhage, or, better still, submerges the apoplectic mass for a time, it will usually be found possible to do so, and even to find the hole in its wall.

(*b*) Aneurism of the branches of the circle of Willis. These are by no means uncommon, and will be considered subsequently.

(*c*) Endarteritis and periarteritis in the cerebral vessels most commonly lead to apoplexy by the production of aneurisms, either miliary or coarse. There are instances in which the most careful search fails to reveal anything but diffuse degeneration of the cerebral vessels, particularly of the smaller branches; so that we must conclude that spontaneous rupture may occur without the previous formation of aneurism.

(*d*) Increased permeability of the walls of the vessels may account for hæmorrhages by *diapedesis* without actual rupture. Such hæmorrhages are not uncommon in cases of contracted kidney, grave anæmia, and various infections and intoxications.

(*e*) In persons over sixty the hemiplegia may depend upon small areas of softening in the gray matter—the *lacunæ* of Marie—areas varying in size from a pin's head to a pea or a small bean, grayish red in tint. The lenticular nucleus is particularly apt to be involved. The blood vessels are always diseased.

The hæmorrhage may be meningeal, cerebral, or intraventricular.

Meningeal hæmorrhage may be outside the dura, between this membrane and the bone, or between the dura and arachnoid, or between the arachnoid and the pia mater. The following are the chief causes of this form of hæmorrhage: Fracture of the skull, in which case the blood usually comes from the lacerated meningeal vessels, sometimes from the torn sinuses. In these cases the blood is usually outside the dura or between it and the arachnoid. The next most frequent cause is rupture of aneurisms on the larger cerebral vessels. The blood is usually subarachnoid. An intracerebral hæmorrhage may burst into the meninges. A special form of meningeal hæmorrhage is found in the new-born, associated with injury during birth. And lastly, meningeal hæmorrhage may occur in the constitutional diseases and fevers. The blood may be in a large quantity at the base; in cases of ruptured aneurism, particularly, it may extend into the cord or upon the cortex. Owing to the greater frequency of the aneurisms in the middle cerebral vessels, the Sylvian fissures are often distended with blood.

Intracerebral hæmorrhage is most frequent in the neighborhood of the corpus striatum, particularly toward the outer section of the lenticular nucleus. The hæmorrhage may be small and limited to the lenticular body, the thalamus, and the internal capsule, or it may extend to the insula. Hæmorrhages confined to the white matter—the centrum semiovale—are rare. Localized bleeding may occur in the crura or in the pons. Hæmorrhage into the cerebellum is not uncommon, and usually comes from the superior cerebellar artery. The extravasation may be limited to the substance or may rupture into the fourth ventricle.

Ventricular Hæmorrhage.—This is rarely primary, coming from the vessels of the plexuses or of the walls. More often it is secondary, following hæmorrhage into the cerebral substance. It is not infrequent in early life and may occur during birth. Of 94 cases collected by Edward Sanders, 7 occurred during the first year, and 14 under the twentieth year. In the cases which I have seen in adults it has almost always been caused by rupture of a vessel in the neighborhood of the caudate nucleus. The blood may be found in one

ventricle only, but more commonly it is in both lateral ventricles, and may pass into the third ventricle and through the aqueduct of Sylvius into the fourth ventricle, forming a complete mould in blood of the ventricular system. In these cases the clinical picture may be that of "*apoplexie foudroyante.*"

Subsequent Changes.—The blood gradually changes in color, and ultimately the hæmoglobin is converted into the reddish brown hæmatoidin. Inflammation occurs about the apoplectic area, limiting and confining it, and ultimately a definite wall may be produced, inclosing a cyst with fluid contents. In other instances a cyst is not formed, but the connective tissue proliferates and leaves a pigmented scar. In meningeal hæmorrhage the effused blood may be gradually absorbed and leave only a staining of the membranes. In other cases, particularly in infants, when the effusion is cortical and abundant, there may be localized wasting of the convolutions and the production of a cyst in the meninges. Possibly porencephaly may arise in this way. Secondary degeneration follows, varying in character according to the location of the hæmorrhage and the actual damage done by it to nerve cells or their medullated axones. Thus, in persons dying some years after a cerebral apoplexy which has produced hemiplegia (lesion of the motor area in the cortex or of the pyramidal tract leading from it), the degeneration may be traced through the cerebral peduncle, the ventral part of the pons, the pyramids of the medulla, the fibres of the direct pyramidal tract of the cord of the same side, and the fibres of the crossed pyramidal tract on the opposite side. After hæmorrhages in the middle and inferior frontal gyri, there follows degeneration of the frontal cerebro-cortico-pontal path going through the anterior limb of the internal capsule and the medial portion of the basis pedunculi to the nuclei pontis; also degeneration of the fibres connecting the nucleus medialis thalami and the anterior part of the nucleus lateralis thalami with the cortex.

When the temporal gyri or their white matter are destroyed by a hæmorrhage the lateral segment of the basis pedunculi degenerates. Cerebellar hæmorrhage, especially if it injure the nucleus dentatus, may lead to degeneration of the brachium conjunctivum.

There may be slow degeneration in the lemniscus medialis, extending as far as the nuclei on the opposite side of the medulla oblongata, after hæmorrhages in the central gyri, hypothalamic region, or dorsal part of the pons. Hæmorrhages destroying the occipital cortex, or subcortical hæmorrhages injuring the optic radiations, occasion slow degeneration (cellulipetal) of the radiations from the lateral geniculate body, and after a time cause marked atrophy or even disappearance of its ganglion cells.

Symptoms.—These may be divided into primary, or those connected with the onset, and secondary, or those which develop later, after the early manifestations have passed away.

Primary Symptoms.—Premonitory indications are rare. As a rule, the patient is seized while in full health or about the performance of some every day action, occasionally, an action requiring strain or extra exertion. Now and then instances are found in which there are sensations of numbness or tingling or pains in the limbs, or even choreiform movements in the muscles of the opposite side, the so-called prehemiplegic chorea. In other cases temporary

disturbances of vision and of associated movements of the eye-muscles have been noted, but none of the prodromata of apoplexy (the so-called "warnings") are characteristic. The onset of the apoplexy, as the symptoms of cerebral hæmorrhage are usually called, varies greatly. There may be sudden loss of consciousness and complete relaxation of the extremities. In such instances the name *apoplectic stroke* is particularly appropriate. In other cases the onset is more gradual and the loss of consciousness may not occur for a few minutes after the patient has fallen, or after the paralysis of the limbs is manifest. In the typical apoplectic attack the condition is as follows: There is deep unconsciousness; the patient can not be roused. The face is injected, sometimes cyanotic, or of an ashen gray hue. The pupils vary; usually they are dilated, sometimes unequal, and always, in deep coma, inactive. If the hæmorrhage be so located that it can irritate the nucleus of the third nerve the pupils are contracted (hæmorrhages into the pons or ventricles). The respirations are slow, noisy, and accompanied with stertor. Sometimes the Cheyne-Stokes rhythm may be present. The chest movements on the paralyzed side may be restricted, in rare instances on the opposite side. The cheeks are often blown out during expiration, with spluttering of the lips. The pulse is usually full, slow, and of increased tension. The temperature may be normal, but is often found subnormal, and, as in a case reported by Bastian, may sink below 95°. In cases of basal hæmorrhage the temperature, on the other hand, may be high. The urine and fæces are usually passed involuntarily. Convulsions are not common. It may be difficult to decide whether the condition is apoplexy associated with hemiplegia or sudden coma from other causes. An indication of hemiplegia may be discovered in the difference in the tonus of the muscles on the two sides. If the arm or the leg is lifted, it drops "dead" on the affected side, while on the other it falls more slowly. The lack of muscular tone of the paralyzed limb may be determined by inspection. In this condition the muscle mass of the thigh acts like a semi-fluid sac and takes the shape determined by gravity. In a patient lying or sitting on a firm support, the thigh of the paralyzed limb is broadened or flattened, while that on the normal side has a more rounded contour. Rigidity also may be present. In watching the movements of the facial muscles in the stertorous respiration it will be seen that on the paralyzed side the relaxation permits the cheek to be blown out in a more marked manner. The head and eyes may be turned strongly to one side—conjugate deviation. In such an event the turning is *toward* the side of the hæmorrhage.

In other cases, in which the onset is not so abrupt, the patient may not lose consciousness, but in the course of a few hours there is loss of power, unconsciousness gradually develops, and deepens into profound coma. This is sometimes termed ingravescent apoplexy. The attack may occur during sleep. The patient may be found unconscious, or wakes to find that the power is lost on one side. Small hæmorrhages in the territory of the central arteries may cause hemiplegia without loss of consciousness. In old persons the hemiplegia may be slight and follow a transient loss of consciousness, and is usually most marked in the leg, which is dragged. It may be quite slight and difficult to make out. It is associated with other senile changes. This is the form very often due to the presence of lacunar softening.

Usually within forty eight hours after the onset of an attack, sometimes

within from two to six hours, there are febrile reaction and more or less constitutional disturbance associated with inflammatory changes about the hæmorrhage and absorption of the blood. The period of inflammatory reaction may continue for from one week to two months. The patient may die in this reaction, or, if consciousness has been regained, there may be delirium or recurrence of the coma. At this period the so-called early rigidity may develop in the paralyzed limbs. The so-called trophic changes may occur, such as sloughing or the formation of vesicles. The most serious of these is the sloughing eschar of the lower part of the back, or on the paralyzed side, which may appear within forty eight hours of the onset and is usually of grave significance. The congestion at the bases of the lungs so common in apoplexy is regarded by some as a trophic change.

Conjugate Deviation.—In a right hemiplegia the eyes and head may be turned to the left side; that is to say, the eyes look toward the cerebral lesion. This is almost the rule ~~in the conjugate deviation of the head and eyes which occurs~~ early in hemiplegia. When, however, convulsions or spasm occur or the state of so-called early rigidity ~~in hemiplegia~~, the conjugate deviation of the head and eyes may be in the opposite direction; that is to say, the eyes look away from the lesion and the head is rotated toward the convulsed side. This symptom may be associated with cortical lesions, particularly, according to some authors, when in the neighborhood of the supramarginal and angular gyri. It may also occur in a lesion of the internal capsule or in the pons, but in the latter situation the conjugate deviation is the reverse of that which occurs in other cases, as the patient looks away from the lesion, and in spasm or convulsion looks toward the lesion.

Hemiplegia.—In cases in which consciousness is restored and the patient improves, a unilateral paralysis may persist due to the destruction of the motor area or the pyramidal tract in any part of its course. Hemiplegia is complete when it involves face, arm, and leg, or partial when it involves only one or other of these parts. This may be the result of a lesion (*a*) of the motor cortex; (*b*) of the pyramidal fibres in the corona radiata and in the internal capsule; (*c*) of a lesion in the cerebral peduncle; or (*d*) in the pons Varolii. The situation of the lesions and their effects are given in Fig. 18. Hæmorrhage is perhaps the most common cause, but tumors and spots of softening may also induce it. The special details of the hemiplegia may here be considered. The face (except in lesions in the lower part of the pons) is involved on the same side as the arm and leg. This results from the fact that the facial muscles stand in precisely the same relation to the cortical centres as those of the arm and leg, the fibres of the upper motor segment of the facial nerve from the cortex decussating just as do those of the nerves of the limbs. The signs of the facial paralysis are usually well marked. There may be a slight difficulty in elevating the eyebrows or in closing the eye on the paralyzed side, or in rare cases the facial paralysis is complete, but the movements may be present with emotion, as laughing or crying. The facial paralysis is partial, involving only the lower portion of the nerve, so that the orbicularis oculi and the frontalis muscles are much less involved than the lower branch. The hypoglossal nerve also is involved. In consequence, the patient can not put out the tongue straight, but it deviates toward the paralyzed side, inasmuch as the genio-hyo-glossus of the sound side is unopposed. In a few cases the

protrusion is toward the side of the lesion, a fact not easily explained in the present state of our knowledge of the nervous control of the tongue. With right hemiplegia there may be aphasia. Even without marked aphasia difficulty in speaking and slowness are common.

The arm is, as a rule, more completely paralyzed than the leg. The loss of power may be absolute or partial. In severe cases it is at first complete. In others, when the paralysis in the face and arm is complete, that of the leg is only partial. The face and arm may alone be paralyzed, while the leg escapes. Less commonly the leg is more affected than the arm, and the face may be only slightly involved.

Certain muscles escape in hemiplegia, particularly those associated in symmetrical movements, as those of the thorax and abdomen, a fact which Broadbent explains by supposing that as the spinal nuclei controlling these movements on both sides constantly act together they may, by means of this intimate connection, be stimulated by impulses coming from only one side of the brain. Hughlings Jackson pointed out that in quiet respiration the muscles on the paralyzed side acted more strongly than the corresponding muscles, but that in forced respiration the reverse condition was true. The degree of permanent paralysis after a hemiplegic attack varies much in different cases. When the restitution is partial, it is always certain groups of muscles which recover rather than others. Thus in the leg the residual paralysis concerns the flexors of the leg and the dorsal flexors of the foot—i. e., the muscles which are active in the second period of walking, shortening the leg, and bringing it forward while it swings. The muscles which lift the body when the foot rests upon the ground, those used in the first period of walking, include the extensors of the leg and the plantar flexors of the foot. These "lengtheners" of the leg often recover almost completely in cases in which the paralysis is due to lesions of the pyramidal tract. In the arms the residual paralysis usually affects the muscle groups which oppose the thumb, those which rotate the arm outward, and the openers of the hand.

As a rule, there is at first no wasting of the paralyzed limbs.

Crossed Hemiplegia.—A paralysis in which there is loss of function in a cerebral nerve on one side with loss of power (or of sensation) on the opposite side of the body is called a crossed or alternate hemiplegia. It is met with in lesions, commonly hæmorrhage, in the crus, the pons, and the medulla (Figs. 18, 12 and 13).

(*a*) *Crus.*—The bleeding may extend from vessels supplying the corpus striatum, internal capsule, and optic thalamus, or the hæmorrhage may be primarily in the crus. In the classical case of Weber, on section of the lower part of the left crus, an oblong clot 15 mm. in length lay just below the medial and inferior surface. The characteristic features of a lesion in this locality are paralysis of arm, face, and leg of the opposite side, and oculo-motor paralysis of the same side—the syndrome of Weber. Sensory changes may also be present. Hæmorrhage into the tegmentum is not necessarily associated with hemiplegia, but there may be incomplete paralysis of the oculo-motor nerve, with disturbance of sensation and ataxia on the opposite side of the body. The optic tract or the lateral geniculate body lying on the lateral side of the crus may be compressed, in which event there will be hemianopia.

(*b*) *Pons and Medulla.*—Lesions may involve the pyramidal tract and one

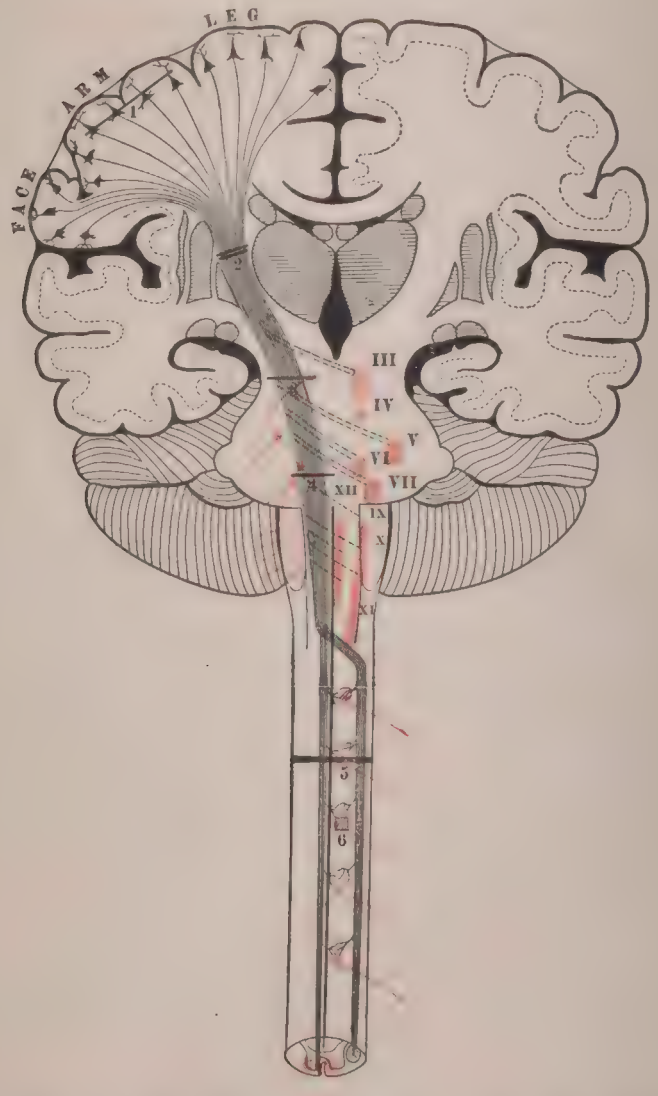


FIG. 18.—DIAGRAM OF MOTOR PATH FROM LEFT BRAIN. The upper segment is black, the lower red. The nuclei of the motor cerebral nerves are shown on the right side; on the left side the cerebral nerves of that side are indicated. A lesion at 1 would cause upper segment paralysis in the arm of the opposite side—cerebral monoplegia; at 2, upper segment paralysis of the whole opposite side of the body—hemiplegia; at 3 (in the crus), upper segment paralysis of the opposite face, arm, and leg, and lower segment paralysis of the eye-muscles on the same side—crossed paralysis; at 4 (in the lower part of the pons), upper segment paralysis of the opposite arm and leg, and lower segment paralysis of the face and external rectus on the same side—crossed paralysis; at 5, upper segment paralysis of all muscles represented below lesion, and lower segment paralysis of muscles represented at level of lesion—spinal paraplegia; at 6, lower segment paralysis of muscles localized at seat of lesion—anterior poliomyelitis. (Van Gehuchten, modified.)

or more of the cerebral nerves. If at the lower aspect of the pons, the facial nerve may be involved, causing paralysis of the face on the same side and hemiplegia on the opposite side. The fifth nerve may be involved, with the fillet (the sensory tract), causing loss of sensation in the area of distribution of the fifth on the same side as the lesion and loss of sensation on the opposite side of the body. The sensory disturbance here is apt to be dissociated, of the syringomyelic type, affecting particularly the sense of pain and temperature.

Sensory Disturbances Resulting from Cerebral Hæmorrhage.—These are variable. Hemianæsthesia may coexist with hemiplegia, but in many instances there is only slight numbing of sensation. When the hemianæsthesia is marked, it is usually the result of a lesion in the internal capsule involving the retrolenticular portion of the posterior limb. In C. L. Dana's study of sensory localization he found that anæsthesia of organic cortical origin was always limited or more pronounced in certain parts, as the face, arm, or leg, and was generally incomplete. Total anæsthesia was either of functional or subcortical origin. Marked anæsthesia was much more common in softening than in hæmorrhage. Complete hemianæsthesia is certainly rare in hæmorrhage. Disturbance of the special senses is not common. Hemianopia may exist on the same side as the paralysis, and there may be diminution in the acuteness of the senses of hearing, taste, and smell. Gowers thinks that homonymous hemianopia of the halves of the visual fields opposite to the lesion is very frequent shortly after the onset, though often overlooked.

Psychic disturbances, variable in nature and degree, may result from cerebral hæmorrhage.

The Reflexes in Apoplectic Cases.—During the apoplectic coma all the reflexes are abolished, but immediately on recovery of consciousness they return, first on the non-hemiplegic side, later, sometimes only after weeks, on the paralyzed side. As to the time of return, especially of the patellar reflexes, marked differences are observable in individual cases. The deep reflexes later are increased on the paralyzed side, and ankle clonus may be present. Plantar stimulation usually gives an extensor response in the great toe (Babinski's reflex). This may occur very early and is an important indication of the paralyzed side. The other superficial reflexes are usually diminished. The sphincters are not affected.

The *course* of the disease depends upon the situation and extent of the lesion. If slight, the hemiplegia may disappear completely within a few days or a few weeks. In severe cases the rule is that the leg gradually recovers before the arm, and the muscles of the shoulder girdle and upper arm before those of the forearm and hand. The face may recover quickly.

Except in the very slight lesions, in which the hemiplegia is transient, changes take place which may be grouped as

Secondary Symptoms.—These correspond to the chronic stage. In a case in which little or no improvement takes place within eight or ten weeks it will be found that the paralyzed limbs undergo certain changes. The leg, as a rule, recovers enough power to enable the patient to get about, although the foot is dragged. Occasionally a recurrence of severe symptoms is seen, even without a new hæmorrhage having taken place. In both arm and leg the condition of *secondary contraction* or *late rigidity* comes on and is always

most marked in the upper extremity. The arm becomes permanently flexed at the elbow and resists all attempts at extension. The wrist is flexed upon the forearm and the fingers upon the hand. The position of the arm and hand is very characteristic. There is frequently, as the contractures develop, a great deal of pain. In the leg the contracture is rarely so extreme. The loss of power is most marked in the muscles of the foot and, to prevent the toes from dragging, the knee in walking is much flexed, or more commonly the foot is swung round in a half circle.

The reflexes are at this stage greatly increased. These contractures are permanent and incurable, and are associated with a secondary descending sclerosis of the motor path. There are instances, however, in which rigidity and contracture do not occur, but the arm remains flaccid, the leg having regained its power. This *hémiplégie flasque* of Bouchard is found most commonly in children. Among other secondary changes in late hemiplegia may be mentioned the following: Tremor of the affected limbs, post-paralytic chorea, the mobile spasm known as athetosis, arthropathies in the joints of the affected side, and muscular atrophy. Athetosis and post-hemiplegic chorea will be considered in the hemiplegia of children. The cool surface and thin glossy skin of a hemiplegic limb are familiar to all. A word may here be said upon the subject of muscular atrophy of cerebral origin.

As a rule, atrophy is not a marked feature in hemiplegia, but in some instances it does occur. It has been thought to be due in some cases to secondary alterations in the gray matter of the ventral horns; but atrophy may follow as a direct result of the cerebral lesion, the ventral horns remaining intact. In Quincke's case atrophy of the arm followed the development of a glioma in the anterior central convolution. The gray matter of the ventral horns was normal. These atrophies are most common in cortical lesions involving the domain of the third main branch of the Sylvian artery, and in central lesions involving the lenticulo-thalamic region. Their explanation is not clear. The wasting of cerebral origin, which occurs most frequently in children, and leads to hemiatrophy of the muscles along with stunted growth of the bones and joints, is to be sharply separated from the hemiatrophy of the muscles of the adult following within a relatively short time upon the hemiplegia.

Diagnosis.—There are three groups of cases which offer increasing difficulty in recognition.

(1) Cases in which the onset is gradual, a day or two elapsing before the paralysis is fully developed and consciousness completely lost, are readily recognized, though it may be difficult to determine whether the lesion is due to thrombosis or to hæmorrhage.

(2) In the sudden apoplectic stroke in which the patient rapidly loses consciousness the difficulty in diagnosis may be still greater, particularly if the patient is in deep coma when first seen.

The first point to be decided is the existence of hemiplegia. This may be difficult, although, as a rule, even in deep coma the limbs on the paralyzed side are more flaccid and drop instantly when lifted; whereas on the non-paralyzed side the muscles retain some degree of tonus. The reflexes may be decreased or lost on the affected side ond there may be conjugate deviation of the head and eyes. Rigidity in the limbs on one side is in favor of a

hemiplegic lesion. It is practically impossible in a majority of these cases to say whether the lesion is due to hæmorrhage, embolism, or thrombosis.

(3) Large hæmorrhage into the ventricles or into the pons may produce sudden loss of consciousness with complete relaxation, so that the condition may simulate coma from uræmia, diabetes, alcoholism, opium poisoning, or epilepsy.

The previous history and the mode of onset may give valuable information. In epilepsy convulsions have preceded the coma; in alcoholism there is a history of constant drinking, while in opium poisoning the coma develops more gradually; but in many instances the difficulty is practically very great, and on more than one occasion I have seen mortifying post mortem disclosures under these circumstances. With diabetic coma the breath often smells of acetone. In ventricular hæmorrhage the coma is sudden and comes on rapidly. The hemiplegic symptoms may be transient, quickly giving place to complete relaxation. Convulsions occur in many cases, and may be the very symptom to lead astray—as in a case of ventricular hæmorrhage which occurred in a puerperal patient, in whom, naturally enough, the condition was thought to be uræmic. Rigidity is often present. In hæmorrhage into the pons convulsions are frequent. The pupils may be strongly contracted, conjugate deviation may occur, and the temperature is apt to rise rapidly. The contraction of the pupils in pontine hæmorrhage naturally suggests opium poisoning. The difference in temperature in the two conditions is a valuable diagnostic point. The apoplectiform seizures of general paresis have usually been preceded by abnormal mental symptoms, and the associated hemiplegia is seldom permanent.

It may be impossible at first to give a definite diagnosis. In admissions to hospitals or in emergency cases the physician should be particularly careful about the following points: The examination of the head for injury or fracture; the urine should be tested for albumin, examined for sugar, and studied microscopically; a careful examination should be made of the limbs with reference to their degree of relaxation or the presence of rigidity, and the condition of the reflexes; the state of the pupils should be noted and the temperature taken. The odor of the breath (alcohol, acetone, chloroform, etc.) should be remarked. The most serious mistakes are made in the case of patients who are drunk at the time of the attack, a combination by no means uncommon in the class of patients admitted to hospital. Under these circumstances the case may erroneously be looked upon as one of alcoholic coma. It is best to regard each case as serious and to bear in mind that this is a condition in which, above all others, mistakes are common.

Prognosis.—From cortical hæmorrhage, unless very extensive, the recovery may be complete without a trace of contracture. This is more common when the hæmorrhage follows injury than when it results from disease of the arteries. Infantile meningeal hæmorrhage, on the other hand, is a condition which may produce idiocy or spastic diplegia.

Large hæmorrhages into the corona radiata, and especially those which rupture into the ventricles, rapidly prove fatal.

The hemiplegia which follows lesions of the internal capsule, the result of rupture of the lenticulo-striate artery, is usually persistent and followed by contracture. When the retro-lenticular fibres of the internal capsule are

involved there may be hemianæsthesia, and later, especially if the thalamus be implicated, hemichorea or athetosis. In any case of cerebral apoplexy the following symptoms are of grave omen: persistence or deepening of the coma during the second and third day; rapid rise in temperature within the first forty-eight hours after the initial fall. In the reaction which takes place on the second or third day the temperature usually rises, and its gradual fall on the third or fourth day with return of consciousness is a favorable indication. The rapid formation of bed-sores, particularly the malignant decubitus of Charcot, is a fatal indication. The occurrence of albumin and sugar, if abundant, in the urine is an unfavorable symptom.

When consciousness returns and the patient is improving, the question is anxiously asked as to the paralysis. The extent of this can not be determined for some weeks. With slight lesions it may pass off entirely. If persistent at the end of a month some grade of permanent palsy is certain to remain, and gradually the late rigidity supervenes.

5. EMBOLISM AND THROMBOSIS

(*Cerebral Softening*)

Embolism.—The embolus usually enters the carotid, rarely the vertebral artery. In the great majority of cases it comes from the left heart and is either a vegetation of a fresh endocarditis or, more commonly, of a recurring endocarditis, or from the segments involved in an ulcerative process. Less often the embolus is a portion of a clot which has formed in the auricular appendix. Portions of clot from an aneurism, thrombi from atheroma of the aorta, or from the territory of the pulmonary veins, may also cause blocking of the branches of the circle of Willis. In the puerperal condition cerebral embolism is not infrequent. It may occur in women with heart disease, but in other instances the heart is uninvolved, and the condition has been thought to be associated with the development of heart clots, owing to increased coagulability of the blood. A majority of cases of embolism occur in heart disease, 89 per cent. (Saveliew). Cases are rare in the acute endocarditis of rheumatic fever, chorea, and febrile conditions. It is much more common in the secondary recurring endocarditis which attacks old sclerotic valves. The embolus most frequently passes to the left middle cerebral artery, as it enters the left carotid oftener than the right because of the more direct course of the blood in the former. The posterior cerebral and the vertebral are less often affected. A large plug may lodge at the bifurcation of the basilar. Embolism of the cerebellar vessels is rare.

Embolism occurs more frequently in women, owing, no doubt, to the greater frequency of mitral stenosis. Contrary to this general statement, Newton Pitt's statistics of 79 cases at Guy's Hospital indicate, however, that males are more frequently affected; as in this series there were 44 males and 35 females. Saveliew gives 54 per cent. in women.

Thrombosis.—Clotting of blood in the cerebral vessels occurs (1) about an embolus, (2) as the result of a lesion of the arterial wall (either endarteritis with or without atheroma or, particularly, the syphilitic arteritis), (3) in aneurisms, both ~~coarse~~ and miliary, and (4) ~~very rarely~~ as a direct result of abnormal conditions of the blood. Thrombosis occasionally follows ligation

of the carotid artery. The thrombosis is most common in the middle cerebral and in the basilar arteries. According to Koliske, softening of limited areas, sufficient to induce hemiplegia, may be caused by sudden collapse of certain cerebral arteries from cardiac weakness.

Anatomical Changes.—Degeneration and softening of the territory supplied by the vessels are the ultimate result in both embolism and thrombosis. Blocking in a terminal artery may be followed by infarction, in which the territory may either be deeply infiltrated with blood (hæmorrhagic infarction) or be simply pale, swollen, and necrotic (anæmic infarction). Gradually the process of softening proceeds, the tissue is infiltrated with serum and is moist, the nerve fibres degenerate and become fatty. The neuroglia is swollen and œdematous. The color of the softened area depends upon the amount of blood. The hæmoglobin undergoes gradual transformation, and the early red color may give place to yellow. Formerly much stress was laid upon the difference between *red*, *yellow*, and *white* softening. The red and yellow are seen chiefly on the cortex. Sometimes the red softening is particularly marked in cases of embolism and in the neighborhood of tumors. The gray matter shows many punctiform hæmorrhages—capillary apoplexy. There is a variety of yellow softening—the *plaques jaunes*—common in elderly persons, which occurs in the gray matter of the convolutions. The spots are from 1 to 2 cm. in diameter, sometimes are angular in shape, the edges cleanly cut, and the softened area is represented by either a turbid, yellow material, or in some instances there is a space crossed by fine trabeculæ, in the meshes of which there is fluid. White softening occurs most frequently in the white matter, and is seen best about tumors and abscesses. Inflammatory changes are common in and about the softened areas. When the embolus is derived from an infected focus, as in ulcerative endocarditis, suppuration may follow. The final changes vary very much. The degenerated and dead tissue elements are gradually but slowly removed, and if the region is small may be replaced by a growth of connective tissue and the formation of a scar. If large, the resorption results in the formation of a cyst. It is surprising for how long an area of softening may persist without much change.

The position and extent of the softening depend upon the obstructed artery. An embolus which blocks the middle cerebral at its origin involves not only the arteries to the anterior perforated space, but also the cortical branches, and in such a case there is softening in the neighborhood of the corpus striatum, as well as in part of the region supplied by the cortical vessels. The freedom of anastomosis between these branches varies a good deal. Thus, there are instances of embolism of the middle cerebral artery in which the softening has involved only the territory of the central branches, in which case blood has reached the cortex through the anterior and posterior cerebrals. When the middle cerebral is blocked (as is perhaps oftenest the case) beyond the point of origin of the central arteries, one or other of its branches is usually most involved. The embolus may lodge in the vessel passing to the third frontal convolution, or in the artery of the ascending frontal or ascending parietal; or it may lodge in the branch passing to the supramarginal and angular gyri, or it may enter the lowest branch which is distributed to the upper convolutions of the temporal lobe. These are practically terminal arteries, and instances frequently occur of softening limited to a part, at any rate, of the

territory supplied by them. Some of the most accurate focalizing lesions are produced in this way.

Symptoms.—Extensive thrombotic softening may exist without any symptoms. It is not uncommon in the post mortem examination of the bodies of elderly persons to find the *plaques jaunes* scattered over the convolutions. So, too, softening may take place in the "silent" regions, as they are termed, without exciting any symptoms. When the central or cortical branches of the middle cerebral arteries are involved the symptoms are similar to those of hæmorrhage from the same arteries. Permanent or transient hemiplegia results. When the central arteries are involved the softening in the internal capsule is commonly followed by permanent hemiplegia. There are certain peculiarities associated with embolism and with thrombosis respectively.

In *embolism* the patient is usually the subject of heart trouble, or there exist some of the conditions already mentioned. The onset is sudden, without premonitory symptoms but sometimes with intense headache. When the embolus blocks the left middle cerebral artery the hemiplegia is associated with aphasia. In *thrombosis*, on the other hand, the onset is more gradual; the patient has previously complained of headache, vertigo, tingling in the fingers; the speech may have been embarrassed for some days; the patient has had loss of memory or is incoherent, or paralysis begins at one part, as the hand, and extends slowly, and the hemiplegia may be incomplete or variable. Abrupt loss of consciousness is much less common, and when the lesion is small consciousness is retained. Thus, in thrombosis due to syphilitic disease, the hemiplegia may come on gradually without the slightest disturbance of consciousness.

The hemiplegia following thrombosis or embolism has practically the characteristics, both primary and secondary, described under hæmorrhage.

The following may be the effects of blocking the different vessels: (*a*) *Vertebral.*—The left branch is more frequently plugged. The effects are involvement of the nuclei in the medulla and symptoms of acute bulbar paralysis. It rarely occurs alone; more commonly with

(*b*) Blocking of the *basilar artery.* When this is entirely occluded, there may be bilateral paralysis from involvement of both motor paths. Bulbar symptoms may be present; rigidity or spasm may occur. The temperature may rise rapidly. The symptoms, in fact, are those of apoplexy of the pons.

(*c*) The *posterior cerebral* supplies the occipital lobe on its medial surface and the greater part of the temporo-sphenoidal lobe. If the main stem be thrombosed there is hemianopia with sensory aphasia. Localized areas of softening may exist without symptoms. Blocking of the main occipital branch (arteria occipitalis of Duret), or of the arteria calcarina, passing to the cuneus may be followed by hemianopia. Hemianæsthesia may result from involvement of the posterior part of the internal capsule. Not infrequently symmetrical thrombosis of the occipital arteries of the two sides occurs, as in Förster's well-known case. Still more frequent is the occurrence of thrombosis of a branch of the posterior cerebral of one hemisphere and a branch of the middle cerebral of the other. It is in such cases that the most pronounced instances of apraxia are met with.

(*d*) *Internal Carotid.*—The symptoms are variable. As is well known, the vessel is in a majority of cases ligated without risk. In other instances tran-

sient hemiplegia follows; in others again the hemiplegia is permanent. These variations depend on the anastomoses in the circle of Willis. If these are large and free, no paralysis follows, but in cases in which the posterior communicating and the anterior communicating vessels are small or absent the paralysis may persist. In No. 7 of my Elwyn series of cases of infantile hemiplegia, the woman, aged twenty four, when six years old, had the right carotid ligated for abscess following scarlet fever, with the result of permanent hemiplegia. Blocking of the internal carotid within the skull by thrombosis or embolism is followed by hemiplegia, coma, and usually death. The clot is rarely confined to the carotid itself, but spreads into its branches and may involve the ophthalmic artery.

(*e*) *Middle Cerebral.*—This is the vessel most commonly involved, and, as already mentioned, if plugged before the central arteries are given off, permanent hemiplegia usually follows from softening of the internal capsule. Blocking of the branches beyond this point may be followed by hemiplegia, which is more likely to be transient, involves chiefly the arm and face, and if the lesion be on the left side is associated with aphasia. There may be plugging of the individual branches passing to the inferior frontal (producing typical motor aphasia if the disease be on the left side), to the anterior and posterior central gyri (usually causing total hemiplegia), to the supramarginal and angular gyri (giving rise, if the thrombosis be on the left side, probably without exception to the so-called visual aphasia (alexia), usually also to right-sided hemionopsia), or to the temporal gyri (in which event with left-sided thrombosis word-deafness results).

(*f*) *Anterior Cerebral.*—No symptoms may follow, and even when the branches which supply the paracentral lobule and the top of the ascending convolutions are plugged the branches from the middle cerebral are usually able to effect a collateral circulation in these parts. Monoplegia of the leg may, however, result. Hebetude and dullness of intellect may occur with obstruction of the vessel.

There is unquestionably greater freedom of communication in the cortical branches of the different arteries than is usually admitted, although it is not possible, for example, to inject the posterior cerebral through the middle cerebral, or the middle cerebral from the anterior; but the absence of softening in some instances in which smaller branches are blocked shows how complete may be the compensation, probably by way of the capillaries. The dilatation of the collateral branches may take place very rapidly; thus a patient with chronic nephritis died about twenty four hours after the hemiplegic attack. There were recent vegetations on the mitral valve and an embolus in the right middle cerebral artery just beyond the first two branches. The central portion of the hemisphere was swollen and œdematous. The right anterior cerebral was greatly dilated, and by measurement its diameter was found to be nearly three times that of the left.

Treatment of Cerebral Hæmorrhage and of Softening.—The chief difficulty in deciding upon a method of treatment is to determine whether the apoplexy is due to hæmorrhage or to thrombosis or embolism. The patient should be placed in bed, with his head moderately elevated and the neck free. He should be kept absolutely quiet. If there are dyspnœa, stertor, and signs of mechanical obstruction to respiration, he should be turned on his side, as rec-

ommended by Bowles. This procedure also lessens the liability to congestion of the lungs. If the signs of intracranial hæmorrhage are certain, and if the arterial tension is high, measures may be taken for its reduction. Of these the most rapid and satisfactory is venesection, which in many cases seems to do good. However, as Cushing has shown experimentally, a rapid and increasing rise of arterial tension usually indicates an endeavor of the vasomotor centres to counteract an increasing intracranial pressure, in this case due to a continuing hæmorrhage. The indication under these circumstances is the relief of the intracranial pressure by craniotomy and removal of the clot, if this is possible. This is particularly applicable in subdural hæmorrhage. Horsley and Spencer, have recently, on experimental grounds, recommended the practice, formerly employed empirically, of compression of the carotid, particularly in the ingravescent form; or even, in suitable cases, passing a ligature round the vessel. An ice-bag may be placed on the head and hot bottles to the feet. The bowels should be freely opened, either by calomel, elaterium, or elaterin. Counter-irritation to the neck or to the feet is not necessary. Catheterization of the bladder may be necessary, especially if the patient remains long unconscious.

Special care should be taken to avoid bed-sores; and if bottles are used to the feet, they should not be too hot, since blisters may be readily caused by a much lower temperature than in health. In the fever of reaction aconite may be indicated, but should be cautiously used. Stimulants are not necessary, unless the pulse becomes feeble and signs of collapse supervene. No digitalis is to be given. During recovery the patient should be still kept entirely at rest, even in the mildest attacks remaining in bed for at least fourteen days. The ice-bag should still be kept to the head. The diet should be light and no medicine other than some placebo should be administered, at least during the first month after the hæmorrhage. Attention should be paid to the position occupied by the paralyzed limb or limbs, which if swollen may be wrapped in cotton batting or flannel.

The treatment of *softening* from thrombosis or embolism is very unsatisfactory. Venesection is not indicated, as it lowers the tension and rather promotes clotting. If, as is often the case, the heart's action is feeble and irregular, stimulants and small doses of digitalis may be given with, if necessary, ether or ammonia. The bowels should be kept open, but it is not well to purge actively, as in hæmorrhage.

In the thrombosis which follows syphilitic disease of the arteries, and which is met with most frequently in men between twenty and forty (in whom the hemiplegia often sets in without loss of consciousness), the iodide of potassium should be freely used, giving from 20 to 30 grains (1.3 to 2 gm.) three times a day, or, if necessary, larger doses. If the syphilis has been recent, mercurials by inunction are also indicated. Practically these are the only cases of hemiplegia in which we see satisfactory results from treatment.

Very little can be done for the hemiplegia which remains. The damage is too often irreparable and permanent, and it is very improbable that iodide of potassium, or any other remedy, hastens in the slightest degree Nature's dealing with the blood clot.

The paralyzed limbs may be gently rubbed once or twice a day, and this should be systematically carried out, in order to maintain the nutrition of the

muscles and to prevent, if possible, contractures. The massage should not, however, be begun until at least ten days after the attack. The rubbing should be *toward* the body, and should not be continued for more than fifteen minutes at a time. After the lapse of a fortnight, or in severe cases a month, the muscles may be stimulated by the faradic current; faradic stimulation alternating with massage, especially if applied to the antagonists of the muscles which ordinarily undergo contracture, is of very great service, even in cases where there can be but little hope of any return of voluntary movement. When contractures occur, electricity properly applied at intervals may still be of some benefit along with the passive movements and frictions, and it has been suggested that tendon transplantation, or indeed cross suture of nerves, may cause some improvement.

In a case of complete hemiplegia the friends should at the outset be frankly told that the chances of full recovery are slight. Power is usually restored in the leg sufficient to enable the patient to get about, but in the majority of instances the finer movements of the hand are permanently lost. The general health should be looked after, the bowels regulated, and the secretions of the skin and kidneys kept active. In permanent hemiplegia in persons above the middle period of life, more or less mental weakness is apt to follow the attack, and the patient may become irritable and emotional.

And, lastly, when hemiplegia has persisted for more than three months and contractures have developed, it is the duty of the physician to explain to the patient, or to his friends, that the condition is past relief, that medicines and electricity will do no good, and that there is no possible hope of cure.

6. ANEURISM OF THE CEREBRAL ARTERIES

Miliary aneurisms are not included, but reference is made only to aneurism of the larger branches. The condition is not uncommon. There were 12 instances in ~~my first~~ 800 autopsies in Montreal. This is a considerably larger proportion than in Newton Pitt's collection from Guy's Hospital, 19 times in 9,000 inspections.

Etiology.—Males are more frequently affected than females. Of ~~my~~ 12 cases, 7 were males. The disease is most common at the middle period of life. One of ~~my~~ cases was a lad of six. Pitt describes one at the same age. The chief causes are (*a*) endarteritis, either simple or syphilitic, which leads to weakness of the wall and dilatation; and (*b*) embolism. These aneurisms are often found with endocarditis. Pitt, in his study of the subject, concludes that it is exceptional to find cerebral aneurism unassociated with fungating endocarditis. The embolus disappears, and dilatation follows the secondary inflammatory changes in the coats of the vessel.

Morbid Anatomy.—The middle cerebral branches are most frequently involved. In ~~my~~ 12 cases the distribution on the arteries was as follows: Internal carotid, 1; middle cerebral, 5; basilar, 3; anterior communicating, 3. Except in one case they were saccular and communicated with the lumen of the vessel by an orifice smaller than the circumference of the sac. In the 154 cases which make up the statistics of Lebert, Durand, and Bartholow the middle cerebral was involved in 44, the basilar in 41, internal carotid in 23, anterior cerebral in 14, posterior communicating in 8, anterior communicating in 8,

vertebral in 7, posterior cerebral in 6, inferior cerebellar in 3 (Gowers). The size of the aneurism varies from that of a pea to that of a walnut. The hæmorrhage may be entirely meningeal with very slight laceration of the brain substance, but the bleeding may be, as Coats has shown, entirely within the substance.

Symptoms.—The aneurism may attain considerable size and cause no symptoms. In a majority of the cases the first intimation is the rupture and the fatal apoplexy. Distinct symptoms are most frequently caused by aneurism of the internal carotid, which may compress the optic nerve or the commissure, causing neuritis or paralysis of the third nerve. A murmur may be audible on auscultation of the skull. Aneurism in this situation may give rise to irritative and pressure symptoms at the base of the brain or to hemianopia. In the remarkable case reported by Weir Mitchell and Dercum an aneurism compressed the chiasma and produced bilateral temporal hemianopia.

Aneurism of the vertebral or of the basilar may involve the nerves from the fifth to the twelfth. A large sac at the termination of the basilar may compress the third nerves or the crura.

The diagnosis is, as a rule, impossible. The larger sacs produce the symptoms of tumor, and their rupture is usually fatal.

7. ENDARTERITIS

In no group of vessels do we more frequently see chronic degenerative changes than in those of the circle of Willis. The condition occurs as:

Arterio-sclerosis, producing localized or diffused thickening of the intima with the formation of atheromatous patches or areas of calcification. In the later stages, as seen in elderly people, the arteries of the circle of Willis may be dilated, stiff, or almost universally calcified.

Syphilitic Endarteritis.—As already mentioned under the section of syphilis, gummatous endarteritis is specially prone to attack the cerebral vessels. It has in itself no specific characters—that is to say, it is impossible in given sections to pick out an endarteritis syphilitica from an ordinary endarteritis obliterans. On the other hand, as already stated, the nodular periarteritis is never seen except in syphilis.

8. THROMBOSIS OF THE CEREBRAL SINUSES AND VEINS

The condition may be primary or secondary. Lebert (1854) and Tonnele were among the first to recognize the condition clinically.

Primary thrombosis of the sinuses and veins is rare. It occurs (*a*) in children, particularly during the first six months of life, usually in connection with diarrhœa. Gowers believes that it is of frequent occurrence, and that thrombosis of the veins is not an uncommon cause of infantile hemiplegia.

(*b*) In connection with chlorosis and anæmia, the so-called *autochthonous sinus-thrombosis.* Of 82 cases of thrombosis in chlorosis, 78 were in the veins and 32 in the cerebral sinuses. The longitudinal sinus is most frequently involved. The thrombosis is usually associated with venous thromboses in other parts of the body, and the patients die, as a rule, in from one to three weeks, but both Bristowe and Buzzard report recoveries.

(*c*) In the terminal stages of cancer, phthisis, and other chronic diseases thrombosis may gradually occur in the sinuses and cortical veins. To the coagulum developing in these conditions the term marantic thrombus is applied.

Secondary thrombosis is much more frequent and follows extension of inflammation from contiguous parts to the sinus wall. The common causes are disease of the internal ear, fracture, compression of the sinuses by tumor, or suppurative disease outside the skull, particularly erysipelas, carbuncle, and parotitis. In secondary cases the lateral sinus is most frequently involved. Of 57 fatal cases in which ear disease caused death with cerebral lesions, there were 22 in which thrombosis existed in the lateral sinuses (Pitt). Tuberculous caries of the temporal bone is often directly responsible. The thrombus may be small, or may fill the entire sinus and extend into the internal jugular vein. In more than one half of these instances the thrombus was suppurating. The disease spreads directly from the necrosis on the posterior wall of the tympanum, ~~According to Voltolini, the inflammation extends~~ by way of the petroso-mastoid canal. It is not so common in disease of the mastoid cells.

Symptoms.—*Primary thrombosis* of the longitudinal sinus may occur without exciting symptoms and is found accidentally at the post mortem. There may be mental dullness with headache. Convulsions and vomiting may occur. In other instances there is nothing distinctive. In the chlorosis cases the head symptoms have, as a rule, been marked. Ball's patient was dull and stupid, had vomiting, dilatation of the pupils, and double choked disks. Slight paresis of the left side occurred. An interesting feature in this case was the development of swelling of the left leg. In the cases reported by Andrews, Church, Tuckwell, Isambard Owen, and Wilks the patients had headache, vomiting, and delirium. Paralysis was not present. In Douglas Powell's case, with similar symptoms, there was loss of power on the left side. Bristowe reports a case of great interest in an anæmic girl of nineteen, who had convulsions, drowsiness, and vomiting. Tenderness and swelling developed in the position of the right internal jugular vein, and a few days later on the opposite side. The diagnosis was rendered definite by the occurrence of phlebitis in the veins of the right leg. The patient recovered.

The onset of such symptoms as have been mentioned in an anæmic or chlorotic girl should lead to the suspicion of cerebral thrombosis. In infants the diagnosis can rarely be made. Involvement of the cavernous sinus may cause œdema about the eyelids or prominence of the eyes.

In the *secondary thrombi* the symptoms are commonly those of septicæmia. For instance, in over 70 per cent. of Pitt's cases the mode of death was by pulmonary pyæmia. This author draws the following important conclusions: (1) The disease spreads oftener from the posterior wall of the middle ear than from the mastoid cells. (2) The otorrhœa is generally of some standing, but not always. (3) The onset is sudden, the chief symptoms being pyrexia, rigors, pains in the occipital region and in the neck, associated with a septicæmic condition. (4) Well-marked optic neuritis may be present. (5) The appearance of acute local pulmonary mischief or of distant suppuration is almost conclusive of thrombosis. (6) The average

duration is about three weeks, and death is generally from pulmonary pyæmia. The chief points in the diagnosis may be gathered from these statements.

Associated with thrombosis of the lateral sinus there may be venous stasis and painful œdema behind the ear and in the neck. The external jugular vein on the diseased side may be less distended than on the opposite side, since owing to the thrombus in the lateral sinus the internal jugular vein is less full than on the normal side, and the blood from the external jugular can flow more easily into it.

Treatment.—In marantic individuals roborants and stimulants are indicated. The position assumed in bed should favor both the arterial and venous circulation. The clothing should not restrict the neck, and care should be taken to avoid *bending* of the neck. The internal administration of potassium iodide and calomel has been recommended in the autochthonous forms, but no treatment is likely to be of any avail.

The secondary forms, especially those following upon disease of the middle ear, are often amenable to operation, and, especially recently, many lives have been saved by surgical intervention after extensive sinus thrombosis.

9. HEMIPLEGIA IN CHILDREN

Etiology.—Of 135 cases, 60 were in boys and 75 in girls. Right hemiplegia occurred in 79, left in 56. In 15 cases the condition was said to be congenital.

In a great majority the disease sets in during the first or second year; thus of the total number of cases, 95 were under two. Cases above the fifth year are rare, only 10 in my series. Neither alcoholism nor syphilis in the parents appears to play an important rôle in this affection. Difficult or abnormal labor is responsible for certain of the cases, particularly injury with the *forceps*. Trauma, such as falls or puncturing wounds, is more rare. The condition followed ligation of the common carotid in one case.

Infectious diseases. All the authors lay special stress upon this factor. In 19 cases in my series the disease came on during or just after one of the specific fevers. I saw one case in which during the height of vaccination convulsions occurred, followed by hemiplegia. The organism of anterior poliomyelitis (Heine-Medin disease) is probably responsible for a considerable share of the cases. In a great majority the disease sets in with a convulsion, in which the child may remain for several hours or longer, and after recovery the paralysis is noticed.

Morbid Anatomy.—In an analysis which I have made of 90 autopsies reported in the literature, the lesions may be grouped under three headings:

(*a*) Embolism, thrombosis, and hæmorrhage, comprising 16 cases, in 7 of which there was blocking of a Sylvian artery, and in 9 hæmorrhage. A striking feature in this group is the advanced age of onset. Ten of the cases occurred in children over six years old.

(*b*) Atrophy and sclerosis, comprising 50 cases. The wasting is either of groups of convolutions, an entire lobe, or the whole hemisphere. The meninges are usually closely adherent over the affected region, though sometimes they look normal. The convolutions are atrophied, firm, and hard, contrasting strongly with the normal gyri. The sclerosis may be diffuse and widespread

over a hemisphere, or there may be nodular projections—the hypertrophic sclerosis. Some of the cases show remarkable unilateral atrophy of the hemisphere. In one of my cases the atrophied hemisphere weighed 169 grams and the normal one 653 grams. The brain tissue may be a mere shell over a dilated ventricle.

(*c*) Porencephalus, which was present in 24 of the 90 autopsies. This term was applied by Heschel (1868) to a loss of substance in the form of cavities and cysts at the surface of the brain, either opening into and bounded by the arachnoid, and even passing deeply into the hemisphere, or reaching to the ventricle. In the study by Audrey of 103 cases of porencephalus, hemiplegia was mentioned in 68 cases.

Practically, then, in infantile hemiplegia cortical sclerosis and porencephalus are the important anatomical conditions. The primary change in the majority of these cases is still unknown. Porencephalus may result from a defect in development or from hæmorrhage at birth. The etiology is clear in the limited number of cases of hæmorrhage, embolism, and thrombosis, but there remains the large group in which the final change is sclerosis and atrophy. What is the primary lesion in these instances? The clinical history shows that in nearly all these cases the onset is sudden, with convulsions—often with slight fever. Strümpell believes that this condition is due to an inflammation of the gray matter—poliencephalitis. A certain number of the cases represent the cerebral sporadic form of epidemic polio-myeloencephalitis. The clinical picture of cases described in the Swedish and other epidemics is identical with that with which we are only too familiar in this disease.

Symptoms.—(*a*) The Onset.—The disease may set in suddenly without spasms or loss of consciousness. In more than half the cases the child is attacked with partial or general convulsions and loss of consciousness, which may last from a few hours to many days. This is one of the most striking features in the disease. Fever is usually present. The hemiplegia, noticed as the child recovers consciousness, is generally complete. Sometimes the paralysis is not complete at first, but occurs after subsequent convulsions. The right side is more frequently affected than the left. The face is commonly not involved.

(*b*) Residual Symptoms.—In some cases the paralysis gradually disappears and leaves scarcely a trace as the child grows up. The leg, as a rule, recovers more rapidly and more fully than the arm, and the paralysis may be scarcely noticeable. In a majority of cases, however, there is a characteristic hemiplegic gait. The paralysis is most marked in the arm, which is usually wasted; the forearm is flexed at right angles, the hand is flexed, and the fingers are contracted. Motion may be almost completely lost; in other instances the arm can be lifted above the head. Late rigidity, which almost always develops, is the symptom which suggested the name *hemiplegia spastica cerebralis* to Heine, the orthopædic surgeon, who first accurately described these cases. It is, however, not constant. The limbs may be quite relaxed even years after the onset. The reflexes are usually increased. In several instances, however, I have known them to be absent. Sensation, as a rule, is not disturbed.

Aphasia is a not uncommon symptom, and occurred in 16 cases of my series.

Mental Defects.—One of the most serious consequences of infantile hemi

plegia is the failure of mental development. A considerable number of these cases drift into the institutions for feeble minded children. Three grades may be distinguished—idiocy, which is most common when the hemiplegia has existed from birth; imbecility, which often increases with the development of epilepsy; and feeble mindedness, a retarded rather than an arrested development.

Epilepsy.—Of the cases in my series, 41 were subjects of convulsive seizures, one of the most distressing sequels of the disease. The seizures may be either transient attacks of *petit mal*, true Jacksonian fits, beginning in and confined to the affected side, or general convulsions.

Post-hemiplegic Movements.—It was in cases of this sort that Weir Mitchell first described the post-hemiplegic movements. They are extremely common, and were present in 34 of my series. There may be either slight tremor in the affected muscles, or incoördinate choreiform movements—the so-called post-hemiplegic chorea—or, lastly,

Athetosis.—In this condition, described by Hammond, there are remarkable spasms of the paralyzed extremities, chiefly of the fingers and toes, and in rare instances of the muscles of the mouth. The movements are involuntary and somewhat rhythmical; in the hand, movements of adduction or abduction and of supination and pronation follow each other in orderly sequence. There may be hyperextension of the fingers, during which they are spread wide apart. This condition is much more frequent in children than in adults. In the latter it may be combined with hemianæsthesia, and the lesion is not cortical, but basic, in the neighborhood of the thalamus. The movements are sometimes increased by emotion. They usually persist during sleep.

Treatment.—The possibility of injury to the brain in protracted labor and in forceps cases should be borne in mind by the practitioner. The former entails the greater risk. In infantile hemiplegia the physician at the outset sees a case of ordinary convulsions, perhaps more protracted and severe than usual. These should be checked as rapidly as possible by the use of the bromides, the application of cold or heat, and a brisk purge. During convulsions chloroform may be administered with safety even to the youngest children. When the paralysis is established not much can be hoped from medicines. In only rare instances does the paralysis entirely disappear. When the recovery is partial the "residual paralysis" is similar to that seen in other lesions of the upper motor segment. Thus in the lower extremity it is the flexors of the leg and the dorsal flexors of the foot which are most often permanently paralyzed (Wernicke). The indications are to favor the natural tendency to improve by maintaining the general nutrition of the child, to lessen the rigidity and contractures by massage and passive motion, and if necessary to correct deformities by mechanical or surgical measures. Much may be done by careful manipulation and rubbing and the application of a proper apparatus. In children the aphasia usually disappears. The epilepsy is a distressing and obstinate symptom, for which a cure can rarely be anticipated. Prolonged periods of quiescence are, however, not uncommon. In the Jacksonian fits the bromides rarely do good, unless there is much irritability and excitement. Operative measures in favorable cases of this particular form of epilepsy may often prove beneficial in reducing the number and severity of the seizures, but it is very unusual for them to be completely or per-

manently checked. The liability to feeble mindedness is the most serious outlook in the infantile cerebral palsies. In many cases the damage is irreparable, and idiocy and imbecility result. With patient training and with care many of the children reach a fair measure of intelligence and self-reliance.

IV. TUMORS, INFECTIONS, GRANULOMATA, AND CYSTS OF THE BRAIN

The following are the most common varieties of new growths within the cranium:

Infectious Granulomata.—(*a*) *Tubercle*, which may form large or small growths, usually multiple. Tuberculosis of the glands or bones may be coexistent, but the tuberculous disease of the brain may occur in the absence of other clinically recognizable tuberculous lesions. The disease is most frequent early in life. Three-fourths of the cases occur under twenty, and one-half of the patients are under ten years of age (Gowers). Of 300 cases of tumor in persons under nineteen collected from various sources by Starr, 152 were tubercle. The nodules are most numerous in the cerebellum and about the base.

(*b*) *Syphiloma* is most commonly found on the cortex cerebri or about the pons. The tumors are superficial, attached to the arteries or the meninges, and rarely grow to a large size, although they may do so. They may be multiple. A gummatous meningitis of the base is common and in this process the oculomotor nerves are often affected. The motor nerves of the eye are particularly prone to syphilitic infiltration, and ptosis and the ordinary forms of squint are common. It is common for the pituitary body to be involved with symptoms suggestive of diabetes insipidus.

Tumors.—(*c*) *Glioma and Neuroglioma.*—These vary greatly in appearance. They may be firm and hard, almost like an area of sclerosis, and are not sharply defined from the surrounding brain substance. They may be soft and very vascular, and hæmorrhages are common. They persist remarkably for many years. Klebs has called attention to the occurrence of elements in them not unlike ganglion-cells. Tumors of this character may contain the "Spinnen" or spider cells; enormous spindle-shaped cells with single large nuclei; cells like the ganglion-cells of nerve-centres with nuclei and one or more processes; and translucent, band-like fibres, tapering at each end, which result from a vitreous or hyaline transformation of the large spindle-cells. A separate type is also recognizable, in which the cells resemble the ependymal epithelium.

(*d*) *Fibrosarcoma* (endothelioma) occurs most commonly in the membranes covering the hemispheres or brain stem, and for a long time may cause injury by its compression effects alone. Tumors of this kind are particularly common in the cerebello-pontine recess. When sarcoma originates in the brain substance it may become one of the largest and most diffusely infiltrating of intracranial growths. When meningeal in origin, it is the form of tumor most amenable to surgical treatment.

(*e*) *Carcinoma* not infrequently is secondary to cancer in other parts. It is seldom primary. Occasionally cancerous tumors have been found in symmetrical parts of the brain.

(*f*) Other varieties occur, such as fibroid growths, which usually develop from the membranes; bony tumors, which grow sometimes from the falx, psammona, cholesteatoma, and angioma. Fatty tumors are occasionally found on the corpus callosum.

Cysts.—These occur between the membranes and the brain, as a result of hæmorrhage or of softening. Porencephalus is a sequel of congenital atrophy or of hæmorrhage, or may be due to a developmental defect. Hydatid cysts have been referred to in the section on parasites. An interesting variety of cyst is that which follows severe injury to the skull in early life. Gliomata often undergo cystic degeneration. Dermoid cyst has been described.

Site.—A majority of all tumors occur in the cerebrum and especially in the centrum ovale. The cerebellum, pons, and membranes are next most often involved. Glioma is more common in the hemispheres and grows slowly. It is usually single. Tubercles are usually multiple. Secondary sarcoma and carcinoma are often mutiple.

Symptoms.—GENERAL.—The following are the most important: *Headache*, either dull, aching, and continuous, or sharp, stabbing, and paroxysmal. It may be diffused over the entire head; sometimes it is limited to the back or front. When in the back of the head it may extend down the neck (especially in tumors in the posterior fossa), and when in the front it may be accompanied with neuralgic pains in the face. Occasionally the pain may be very localized and associated with tenderness on pressure.

Choked disk (optic neuritis) occurs in four fifths of all the cases (Gowers). It should be looked for in every patient presenting cerebral symptoms, for it may be present in high degree without impairment of vision. Loss of visual acuity usually indicates that optic atrophy has set in. It is usually double, but occasionally is found in only one eye. A growth may develop slowly and attain considerable size without producing optic neuritis. On the other hand, it may occur with a very small tumor, when this tumor is so situated as to cause internal hydrocephalus. J. A. Martin, from an extensive analysis of the literature with reference to the localizing value, concludes: When there is a difference in the amount of the neuritis in each eye it is more than twice as probable that the tumor is on the side of the most marked neuritis. It is constant in tumors of the corpora quadrigemina, present in 89 per cent. of cerebellar tumors, and absent in nearly two thirds of the cases of tumor of the pons, medulla, and of the corpus callosum. It is least frequent in cases of tuberculous tumor; most common in cases of glioma and cystic tumors.

Paton and Holmes, who reported upon the eyes of 700 cases of cerebral tumor, concluded that the essential feature of the associated optic neuritis is œdema, and in 60 eyes examined histologically the one unfailing change was acute œdema, the origin of which they attribute to the venous engorgement.

Vomiting is a common feature and, with headache and optic neuritis, makes up the characteristic clinical picture of cerebral tumor. An important point is the absence of definite relation to the meals. A chemical examination shows that the vomiting is independent of digestive disturbances. It may be very obstinate, particularly in growths of the cerebellum and the pons.

Giddiness is often an early symptom. The patient complains of vertigo on rising suddenly or on turning quickly.

Mental Disturbance.—The patient may act in an odd, unnatural manner,

or there may be stupor and heaviness. The patient may become emotional or silly, or there are symptoms resembling hysteria.

Convulsions, either general and resembling true epilepsy or localized (Jacksonian) in character. Seizures beginning with a gustatory or olfactory aura are particularly common with tumors originating in the infundibular region. There may be *slowing of the pulse*, as in all cases of increased intracranial pressure.

Localizing Symptoms.—Focal symptoms often occur, but it must not be forgotten that these may be *indirectly* produced. The smaller the tumor and the less marked the general symptoms of cerebral compression the more likely is it that any focal symptoms occurring are of *direct* origin.

(*a*) *Central Motor Area.*—The symptoms are either irritative or destructive in character. Irritation in the lower third may produce spasm in the muscles of the face, in the angle of the mouth, or in the tongue. The spasm with tingling may be strictly limited to one muscle group before extending to others, and this Seguin terms the *signal symptom*. The middle third of the motor area contains the centres controlling the arm, and here, too, the spasm may begin in the fingers, in the thumb, in the muscles of the wrist, or in the shoulder. In the upper third of the motor areas the irritation may produce spasm beginning in the toes, in the ankles, or in the muscles of the leg. In many instances the patient can determine accurately the point of origin of the spasm, and there are important sensory disturbances, such as numbness and tingling, which may be felt first at the region affected.

In all cases it is important to determine, first, the point of origin, the *signal symptom;* second, the order or march of the spasm; and third, the subsequent condition of the parts first affected, whether it is a state of paresis or anæsthesia.

Destructive lesions in the motor zone cause paralysis, which is often preceded by local convulsive seizures; there may be a monoplegia, as of the leg, and convulsive seizures in the arm, often due to irritation in these centres. Tumors in the neighborhood of the motor area may cause localized spasms and subsequently, as the centres are invaded by the growth, paralysis occurs. When tumors are situated in the left hemisphere the speech mechanism is apt to be involved if the transverse temporal gyrus or the third frontal convolution and their connecting path are implicated.

(*b*) *Prefrontal Region.*—Neither motor nor sensory disturbance may be present. The general symptoms are often well marked. The most striking feature of growths in this region is mental torpor and gradual imbecility. Particularly when the left side is involved mental characteristics may be greatly altered. In its extension downward the tumor may involve on the left side the lower frontal convolution and produce aphasia, or in its progress backward cause irritative or destructive lesions of the motor area. Exophthalmos on the side of the tumor may occur and be helpful in diagnosis.

(*c*) Tumors in the *parieto-occipital lobe*, particularly on the right side, may grow to a large size without causing any symptoms. There may be word-blindness and mind-blindness when the left angular gyrus and its underlying white matter are involved, and paraphasia. Astereognosis may accompany growths in the superior parietal region.

(*d*) Tumors of the *occipital lobe* produce hemianopia, and a bilateral

lesion may produce blindness. Tumors in this region on the left hemisphere may be associated with word-blindness and mind-blindness. In all cases of tumor a careful study should be made of the fields of vision. In addition to the lateral hemianopia there may be remarkable visual hallucinations, and in tumors of the left occipital lobe dissociation of the color sense and inability to find the proper colors of various objects presented.

(*e*) Tumors in the *temporal lobe* may attain a large size without producing symptoms. In their growth they involve the lower motor centres. On the left side involvement of the transverse temporal gyri (auditory sense area) may be associated with word-deafness.

(*f*) Tumors growing in the neighborhood of the *basal ganglia* produce hemiplegia from involvement of the internal capsule. Limited growths in either the nucleus caudatus or the nucleus lentiformis of the corpus striatum do not necessarily cause paralysis. Tumors in the thalamus opticus may also, when small, cause no symptoms, but, increasing, they may involve the fibres of the sensory portion of the internal capsule, producing hemianopia and sometimes hemianæsthesia. Growths in this situation are apt to cause early optic neuritis, and, growing into the third ventricle, may cause a distention of the lateral ventricles. What has been termed the thalamic syndrome may be present—hemianæsthesia to pain, touch and temperature, with the loss of deep sensibility. With this there may be a very remarkable type of pain, involving the hand and arm and the foot and leg, on the affected side, a sense of burning discomfort rather than sharp pain. Ataxic features are usually present and astereognosis. Motor hemiplegia may be present, and it is unaccompanied by contractures (Dana).

Growths in the *corpora quadrigemina* are rarely limited, but most commonly involve the crura cerebri as well. Ocular symptoms are marked. The pupil reflex is lost and there is nystagmus. In the gradual growth the third nerve is involved as it passes through the crus, in which case there will be oculo-motor paralysis on one side and hemiplegia on the other, a combination almost characteristic of unilateral disease of the crus.

(*g*) Tumors of the *pons* and *medulla*. The symptoms are chiefly those of pressure upon the nerves emerging in this region. In disease of the pons the nerves may be involved alone or with the pyramidal tract. Of 52 cases analyzed by Mary Putnam Jacobi, there were 13 in which the cerebral nerves were involved alone, 13 in which the limbs were affected, and 26 in which there were hemiplegia and involvement of the nerves. Twenty-two of the latter had what is known as alternate paralysis—i. e., involvement of the nerves on one side and of the limbs on the opposite side. In 4 cases there were no motor symptoms. In tuberculosis (or syphilis) a growth at the inferior and inner aspects of the crus may cause paralysis of the third nerve on one side, and of the face, tongue, and limbs on the opposite side (syndrome of Weber). A tumor growing in the lower part of the pons usually involves the sixth nerve, producing internal strabismus, the seventh nerve, producing facial paralysis, and the auditory nerve, causing deafness. Conjugate deviation of the eyes to the side opposite that on which there is facial paralysis also occurs. When the motor cerebral nerves are involved the paralyses are of the peripheral type (lower segment paralyses).

Tumors of the *medulla* may involve the cerebral nerves alone or cause

in some instances a combination of hemiplegia with paralysis of the nerves. Paralyses of the nerves are helpful in topical diagnosis, but the fact must not be overlooked that one or more of the cerebral nerves may be paralyzed as a result of a much increased general intracranial pressure. Signs of irritation in the ninth, tenth, and eleventh nerves are usually present, and produce difficulty in swallowing, irregular action of the heart, irregular respiration, vomiting, and sometimes retraction of the head and neck. The hypoglossal nerve is least often affected. The gait may be unsteady or, if there is pressure on the cerebellum, ataxic. Occasionally there are sensory symptoms, numbness, and tingling. Toward the end convulsions may occur.

(*h*) Tumors of the *cerebellum* may be latent, but they usually give rise to very characteristic symptoms, headache in the occipital region, giddiness, incoordination, but there is nothing definite in the direction of the swaying, and early optic neuritis. They may be intracerebellar or extracerebellar.

Tumors or enlargements of the *pituitary gland* itself, or growths from a congenital *anlage* in its neighborhood which implicate the pituitary gland secondarily, are very common. The congenital tumors arise presumably from developmental faults, and show either a teratomatous character or are solid or cystic tumors with squamous epithelium, often attaining adamantine characteristics. The most common tumor is a so-called struma (malignant adenoma) of the gland proper. There are characteristic signs of pressure upon the neighborhood structures, bitemporal hemianopia being a frequent though not invariable feature. These lesions may occur in patients who have suffered from acromegaly, or in those who show signs of glandular deficiency or dyspituitarism, and in whom there may or may not be suggestive acromegalic tendencies. The X-rays are most useful in diagnosis.

Diagnosis.—From the general symptoms alone the existence of tumor may be determined, for the combination of headache, optic neuritis, and vomiting is distinctive. As pointed out by R. T. Williamson, progressive hemiplegia, without other symptoms, a paralysis, which gradually becomes more marked day by day and week by week, is almost pathognomic, even in the absence of optic neuritis, headache, and vomiting. The two exceptions to this rule appear to be in cerebral abscess, and in rare instances a polioencephalitis. It must not be forgotten that severe headache and neuro-retinitis may be caused by Bright's disease. The localization must be gathered from the consideration of the symptoms above detailed and from the data given in the section on Topical Diagnosis of Diseases of the Brain. Mistakes are most likely to occur in connection with uræmia, hysteria, vascular lesions, abscess, serous meningitis, hydrocephalus, and general paralysis; but careful consideration of all the circumstances of the case usually enables the practitioner to avoid error.

Prognosis.—Syphilitic tumors alone are amenable to medical treatment. Tuberculous growths occasionally cease to grow and become calcified. The gliomata and fibromata, particularly when the latter grow from the membranes, may last for years. I have described a case of small, hard glioma, in which the Jacksonian epilepsy persisted for fourteen years. Hughlings Jackson has reported cases of glioma in which the symptoms lasted for over ten years. The more rapidly growing sarcomata usually prove fatal in from six to eighteen months. Death may be sudden, particularly in growths near the medulla;

more commonly it is due to coma in consequence of gradual increase in the intracranial pressure.

Treatment.—(*a*) MEDICAL.—A Wassermann test of the blood and cerebrospinal fluid should always be made before antiluetic measures are instituted. It must not be overlooked that vigorous treatment with potassium iodide often causes a temporary amelioration of pressure symptoms due to a glioma, so that the therapeutic test is not entirely a dependable one. If syphilis is proved the iodide of potassium and mercury should be given. Salvarsan is sometimes given in repeated small doses. Nowhere do we see more brilliant therapeutical effects than in certain cases of cerebral gummata. The iodide should be given in increasing doses. In tuberculous tumors the outlook is less favorable, though instances of cure are reported, and there is post mortem evidence to show that the solitary tuberculous tumors may undergo changes and become obsolete. A general tonic treatment is indicated in these cases. The headache usually demands prompt treatment. The iodide of potassium in full doses sometimes gives marked relief. An ice-cap for the head or, in the occipital headache, the application of the Paquelin cautery may be tried. The bromides are not of much use in the headache from this cause, and, as the last resort, morphia must be given. For the convulsions bromide of potassium is of little service.

(*b*) SURGICAL.—Scores of tumors of the brain have now been successfully removed. Though the percentage of cases in which total enucleation is possible is doubtless small, yet in all cases marked amelioration of the pressure symptoms is possible by modern surgical measures. It is important that they should be instituted early, even in the absence of localizing symptoms, for the sake of preserving vision. The most advantageous cases are the localized fibromata and sarcomata growing from the dura and only compressing the brain substance. Of late years there have been numerous successful operations with removal of growths from the cerebellum and cerebello-pontine recess. The safety with which the exploratory operation can be made warrants it in all doubtful cases. For two objects the so-called decompression operation may be performed, to relieve the headache, which it sometimes does promptly and permanently, and to save sight. It is now very generally practised by surgeons, and the reduction of the greatly increased intracranial pressure may cause the choked disk to subside and the risk of subsequent atrophy is much diminished.

V. INFLAMMATION OF THE BRAIN

1. ACUTE ENCEPHALITIS

A focal or diffuse inflammation of the brain substance, usually of the gray matter (poliencephalitis), is met with (*a*) as a result of trauma; (*b*) in certain intoxications, alcohol, food poisoning, and gas poisoning; (*c*) following the acute infections; and (*d*) as one of the varieties of the poliomyeloencephalitis (Heine-Medin disease). The anatomical features are those of an acute hæmorrhagic poliencephalitis, corresponding in histological details with acute polio-myelitis. Focal forms are seen in ulcerative endocarditis, in which the gray matter may present deeply hæmorrhagic areas, firmer than the sur-

rounding tissue. In the fevers there may be more extensive regions, involving two or three convolutions. This acute hæmorrhagic poliencephalitis superior is thought by Strümpell to be the essential lesion in infantile hemiplegia, and it seems probable that many of the cases represent the sporadic form (cerebral variety of the Heine-Medin disease). Localizing symptoms are usually present, though they may be obscured in the severity of the general infection. The most typical encephalitis accompanies the meningitis in cerebro-spinal fever.

In acute mania, in delirium tremens, in chorea insaniens, in the maniacal form of exophthalmic goitre, and in the so-called cerebral forms of the malignant fevers the gray cortex is deeply congested, moist, and swollen, and with the recent finer methods of research will probably show changes which may be classed as encephalitis.

The *symptoms* are not very definite. In severe forms they are those of an acute infection; some cases have been mistaken for typhoid fever. The onset may be abrupt in an individual apparently healthy. Other cases have occurred in the convalescence from the fevers, particularly influenza. One of J. J. Putnam's cases followed mumps. The general symptoms are those which accompany all severe acute affections of the brain—headache, somnolence, coma, delirium, vomiting, etc. The local symptoms are very varied, depending on the extent of the lesions, and may be irritative or paralytic. Usually fatal within a few weeks, cases may drag on for weeks or months and recover, generally with paralysis.

2. ABSCESS OF THE BRAIN

Definition.—Purulent encephalitis with abscess formation the result of infection by micro-organisms.

Etiology.—Suppuration of the brain substance is rarely if ever primary, but results, as a rule, from extension of inflammation from neighboring parts or infection from a distance through the blood. The question of idiopathic brain abscess need scarcely be considered, though occasionally instances occur in which it is extremely difficult to assign a cause. There are three important etiological factors.

(*a*) *Trauma.* Falls upon the head or blows, with or without abrasion of the skin. More commonly it follows fracture or punctured wounds. In this group meningitis is frequently associated with the abscess. As Bergmann says, simple trauma or concussion can never produce abscess but organisms may enter through a laceration of the base opening one of the many sinuses.

(*b*) By far the most important infective foci are those which arise in *direct extension from disease of the middle ear*, of the *mastoid cells*, or of the *frontal sinuses*. From the roof of the mastoid antrum the infection readily passes to the sigmoid sinus and induces an infective thrombosis. In other instances the dura becomes involved, and a subdural abscess is formed, which may readily involve the arachnoid or the pia mater. In another group the inflammation extends along the lymph spaces, or the thrombosed veins, into the substance of the brain and causes suppuration. Macewen thinks that without local areas of meningitis the infective agents may be carried through the lymph and blood channels into the cerebral substance. Infection which ex-

tends from the roof of the tympanic cavity is most likely to be followed by abscess in the temporal lobe, while infection extending from the mastoid cells causes most frequently sinus thrombosis and cerebellar abscess.

(*c*) *In septic processes.* Abscess of the brain is not often found in pyæmia. In ulcerative endocarditis multiple foci of suppuration are common. Localized bone disease and suppuration in the liver are occasional causes. Certain inflammations in the lungs, particularly bronchiectasis, as already referred to in connection with Schörstein's researches, may be followed by abscess. It is an occasional complication of empyema. Abscess of the brain may follow the specific fevers. Bristowe has called attention to its occurrence as a sequel of influenza. The largest number of cases occur between the twentieth and fortieth years, and the condition is more frequent in men than in women. Holt has collected 25 cases in children under five years of age, the chief causes of which were otitis media and trauma.

Morbid Anatomy.—The abscess may be solitary or multiple, diffuse or circumscribed. Practically any one of the different varieties of pyogenic bacteria may be concerned. The bacteriological examination often shows a mixture of different varieties. Occasionally cultures are sterile, owing to death of the bacteria. In the acute, rapidly fatal cases following injury the suppuration is not limited; but in long standing cases the abscess is inclosed in a definite capsule, which may have a thickness of from 2 to 5 mm. The pus varies much in appearance, depending upon the age of the abscess. In early cases it may be mixed with reddish *débris* and softened brain matter, but in the solitary encapsulated abscess the pus is distinctive, having a greenish tint, an acid reaction, and a peculiar odor, sometimes like that of sulphuretted hydrogen. The brain substance surrounding the abscess is usually œdematous and infiltrated. The size varies from that of a walnut to that of a large orange. There are cases on record in which the cavity has occupied the greater portion of a hemisphere. Multiple abscesses are usually small. In four fifths of all cases the abscess is solitary. Suppuration occurs most frequently in the cerebrum, and the temporal lobe is more often involved than other parts, and always on the side of the ear disease. The cerebellum is the next most common seat, particularly in connection with ear disease.

Symptoms.—Following injury or operation the disease may run an *acute* course, with fever, headache, delirium, vomiting, and rigors. The symptoms are those of suppurative meningo-encephalitis, and it may be very difficult to determine, unless there are localizing symptoms, whether there is really suppuration in the brain substance. In the cases following ear disease the symptoms may at first be those of meningeal irritation. There may be irritability, restlessness, severe headache, and aggravated earache. Other striking symptoms, particularly in the more prolonged cases, are drowsiness, slow cerebration, vomiting, and optic neuritis. In the chronic form of brain abscess which may follow injury, otorrhœa, or local lung trouble, there may be a latent period ranging from one or two weeks to several months, or even a year or more. In the "silent" regions, when the abscess becomes encapsulated there may be no symptoms whatever during the latent period. During all this time the patient may be under careful observation and no suspicion be aroused of the existence of suppuration. Then severe headache, vomiting, and fever set in, perhaps with a chill. So, too, after a blow upon the head or a fracture

the symptoms of the lesion may be transient, and months afterward cerebral symptoms of the most aggravated character may develop.

The localization of the lesion is often difficult. If situated in or near the motor region there may be convulsions or paralysis, and it is to be remembered that an abscess in the temporal lobe may compress the lower part of the pre-central convolution and produce paralysis of the arm and face, and on the left side cause aphasia. A large abscess may exist in the frontal lobe without causing paralysis, but in these cases there is almost always some mental dullness. In the temporal lobe, the common seat, there may be no focalizing symptoms. So also in the parieto-occipital region; though here early examination may lead to the detection of hemianopia. In abscess of the cerebellum vomiting is common. If the middle lobe is affected there may be staggering —cerebellar incoördination. Localizing symptoms in the pons and other parts are still more uncertain.

Diagnosis.—In the acute cases there is rarely any doubt. A consideration of possible etiological factors is of the highest importance. The history of injury followed by fever, marked cerebral symptoms, the onset of rigors, delirium, and perhaps paralysis, make the diagnosis certain. In chronic ear disease, such cerebral symptoms as drowsiness and torpor, with irregular fever, supervening upon the cessation of a discharge, should excite the suspicion of abscess. Cases in which suppurative processes exist in the orbit, nose, or naso-pharynx, or in which there has been subcutaneous phlegmon of the head or neck, a parotitis, a facial erysipelas, or tuberculous or syphilitic disease of the bones of the skull, should be carefully watched, and immediately investigated should cerebral symptoms appear. It is particularly in the chronic cases that difficulties arise. The symptoms resemble those of tumor of the brain; indeed, they are those of tumor plus fever. Choked disk, however, so commonly associated with tumor, is very frequently absent in abscess of the brain. In a patient with a history of trauma or with localized lung or pleural trouble, who for weeks or months has had slight headache or dizziness, the onset of a rapid fever, especially if it be intermittent and associated with rigors, intense headache, and vomiting, points strongly to abscess. The pulse rate in cases of cerebral abscess is usually accelerated, but cases are not rare in which it is slowed. Macewen lays stress upon the value of percussion of the skull as an aid in diagnosis. The note, which is uniformly dull, becomes much more resonant when the lateral ventricles are distended in cerebellar abscess and in conditions in which the venæ Galeni are compressed.

It is not always easy to determine whether the meninges are involved with the abscess. Often in ear disease the condition is that of meningo-encephalitis. Sometimes in association with acute ear disease the symptoms may simulate closely cerebral meningitis or even abscess. Indeed, Gowers states that not only may these general symptoms be produced by ear disease, but even distinct optic neuritis.

Treatment.—A remarkable advance has been made of late years in dealing with these cases, owing to the impunity with which the brain can be explored. In ear disease free discharge of the inflammatory products should be promoted and careful disinfection practiced. The treatment of injuries and fractures comes within the scope of the surgeon. The acute symptoms, such as fever, headache, and delirium, must be treated by rest, an ice-cap, and, if necessary,

local depletion. In all cases, when a reasonable suspicion exists of the occurrence of abscess, the brain should be explored. The cases following ear disease, in which the suppuration is in the temporal lobe or in the cerebellum, offer the most favorable chances of recovery. The localization can rarely be made accurately in these cases, and the operator must be guided more by general anatomical and pathological knowledge. In cases of injury the trephine should be applied over the seat of the blow or the fracture. In ear disease the suppuration is most frequent in the temporal lobe or in the cerebellum, and the operation should be performed at the points most accessible to these regions. Crowe's discovery of the secretion of hexamethylenamine into the cerebro-spinal fluid suggests its administration in every case in which meningeal infection is threatened or has occurred.

VI. HYDROCEPHALUS

Definition.—A condition, congenital or acquired, in which there is a great accumulation of fluid within the ventricles of the brain.

The term hydrocephalus has also been applied to the collection of fluid between the cortex of the brain and the skull known in this situation as *hydrocephalus externus* or *hydrocephalus ex vacuo*, a condition common in cases of atrophy of the brain substance, met with in old age, after hæmorrhages, softenings, or scleroses, in lingering and cachectic diseases, as cancer, chronic nephritis, chronic alcoholism, and sometimes in rickets. Occasionally the disease is caused by meningeal cysts. A true dropsy, however, of the arachnoid sac probably does not occur.

The cases may be divided into three groups—idiopathic internal hydrocephalus (serous meningitis), congenital or infantile, and secondary or acquired.

Serous Meningitis (Quincke) (*Idiopathic Internal Hydrocephalus; Angio-neurotic Hydrocephalus*).—This remarkable form, described by Quincke, is very important, since a knowledge of the condition may explain very anomalous and puzzling cases. It is an ependymitis causing a serous effusion into the ventricles, with distention and pressure effects. It may be compared to the serous exudates in the pleura or in synovial membranes. It is not certain that the process is inflammatory, and Quincke likens it to the angio-neurotic œdema of the skin. In very acute cases the ependyma may be smooth and natural looking; in more chronic cases it may be thickened and sodden. The exudate does not differ from the normal, and if on lumbar puncture a fluid is removed of a specific gravity above 1.009, with albumin above two tenths per cent., the condition is more likely to be hydrocephalus from stasis, secondary to tumor, etc.

Both children and adults are affected, the latter more frequently. In the acute form the condition is mistaken for tuberculous or purulent meningitis. There are headache, retraction of the neck, and signs of increased intracranial pressure, choked disks, slow pulse, etc. Fever is usually absent, but I have seen one case with recurring paroxysms of fever, and Morton Prince has described a similar one. In both the exudate was clear and the ependyma not acutely inflamed. Quincke has reported cases of recovery. In the chronic

form the symptoms are those of tumor—general, such as headache, slight fever, somnolence, and delirium; and local, as exophthalmos, optic neuritis, spasms, and rigidity of muscles and paralysis of the cerebral nerves. Remarkable exacerbations occur, and the symptoms vary in intensity from day to day. Recovery may follow after an illness of many weeks, and some of the reported cases of disappearance of all symptoms of brain tumor belong in this category.

Congenital Hydrocephalus.—The enlarged head may obstruct labor; more frequently the condition is noticed some time after birth. The cause is unknown. It has occurred in several members of the same family.

The anatomical condition in these cases offers no clew to the nature of the trouble. The lateral ventricles are enormously distended, but the ependyma is usually clear, sometimes a little thickened and granular, and the veins large. The choroid plexuses are vascular, sometimes sclerotic, but often natural looking. The third ventricle is enlarged, the aqueduct of Sylvius dilated, and the fourth ventricle may be distended. The quantity of fluid may reach several litres. It is limpid and contains a trace of albumin and salts. The changes in consequence of this enormous ventricular distention are remarkable. The cerebral cortex is greatly stretched, and over the middle region the thickness may amount to no more than a few millimetres without a trace of the sulci or convolutions. The basal ganglia are flattened. The skull enlarges, and the circumference of the head of a child of three or four years may reach 25 or even 30 inches. The sutures widen. Wormian bones develop in them, and the bones of the cranium become exceedingly thin. The veins are marked beneath the skin. A fluctuation wave may sometimes be obtained, and Fisher's brain murmur may be heard. The orbital plates of the frontal bone are depressed, causing exophthalmos, so that the eyeballs can not be covered by the eyelids. The small size of the face, widening somewhat above, is striking in comparison with the enormously expanded skull.

Convulsions may occur. The reflexes are increased, the child learns to walk late, and ultimately in severe cases the legs become feeble and sometimes spastic. Sensation is much less affected than motility. Choked disk is not uncommon. The mental condition is variable; the child may be bright, but, as a rule, there is some grade of imbecility. The congenital cases usually die within the first four or five years. The process may be arrested and the patient may reach adult life. Cases of this sort are not very uncommon. Even when extreme, the mental faculties may be retained, as in Bright's celebrated patient, Cardinal, who lived to the age of twenty-nine, and whose head was translucent when the sun was shining behind him. Care must be taken not to mistake the rachitic head for hydrocephalus.

Acquired Chronic Hydrocephalus.—This is stated to be occasionally primary (idiopathic)—that is to say, it comes on spontaneously in the adult without observable lesion. Dean Swift is said to have died of hydrocephalus, but this seems very unlikely. It is based upon the statement that "he (Mr. Whiteway) opened the skull and found much water in the brain," a condition no doubt of *hydrocephalus ex vacuo*, due to the wasting associated with his prolonged illness and paralysis. In nearly all cases there is either a tumor at the base of the brain or in the third ventricle, which compresses the venæ Galeni. The passage from the third to the fourth ventricle may be closed,

either by a tumor or by parasites. More rarely the foramen of Magendie, through which the ventricles communicate with the cerebro-spinal meninges, becomes closed by meningitis. Chronic inflammations of the ependyma may in similar fashion block the foramina of exit of the ventricular fluid. There may be unilateral hydrocephalus from closure of one of the foramina of Monro. In cerebro-spinal fever, particularly in the sporadic form, the foramina of exit of the fluid may be occluded, with great distention of the ventricles. These conditions, occurring in adults, may produce the most extreme hydrocephalus without any enlargement of the head. Even when the tumor begins early in life there may be no expansion of the skull. In the case of a girl aged sixteen, blind from her third year, the head was not unusually large, the ventricles were enormously distended, and in the Rolandic region the brain substance was only 5 mm. in thickness. A tumor occupied the third ventricle. In a case of cholesteatoma of the floor of the third ventricle, in which the symptoms persisted at intervals for eight or nine years, the ventricles were enormously distended without enlargement of the skull. In other instances the sutures separate and the head gradually enlarges.

The symptoms of hydrocephalus in the adult are curiously variable. In the first case mentioned there were early headaches and gradual blindness; then a prolonged period in which she was able to attend to her studies. Headaches again supervened, the gait became irregular and somewhat ataxic. Death occurred suddenly. In the other case there were prolonged attacks of coma with a slow pulse, and on one occasion the patient remained unconscious for more than three months. Gradually progressing optic neuritis without focalizing symptoms, headache, and attacks of somnolence or coma are suggestive symptoms. These cases of acquired chronic hydrocephalus can not be certainly diagnosed during life, though in certain instances the condition may be suspected. They simulate tumor very closely.

Treatment.—Very little can be done to relieve hydrocephalus. Medicines are powerless to cause the absorption of the fluid. In the meningitis serosa Quincke advises the use of mercury. Many operative procedures have been devised, tapping of the ventricles, lumbar puncture, making communications between the ventricles and the subarachnoid spaces, into the extracranial tissues, or into the retro-peritoneal tissues and through the body of the fifth lumbar vertebra; and Cushing has practiced an anastomosis by means of a transplanted vein between the external jugular and the subdural space. Bramann claims beneficial results from puncture of the corpus callosum.

F. DISEASES OF THE PERIPHERAL NERVES

I. NEURITIS

(*Inflammation of the Bundles of Nerve Fibres*)

Neuritis may be *localized* in a single nerve, or *general*, involving a large number of nerves, in which case it is usually known as *multiple neuritis* or *polyneuritis*.

Etiology.—*Localized neuritis* arises from (*a*) cold, which is a very fre-

quent cause, as, for example, in the facial nerve. This is sometimes known as rheumatic neuritis. (*b*) Traumatism—wounds, blows, direct pressure on the nerves, the tearing and stretching which follow a dislocation or a fracture, and the hypodermic injection of ether. Under this section come also the professional palsies, due to pressure in the exercise of certain occupations. (*c*) Extension of inflammation from neighboring parts, as in a neuritis of the facial nerve due to caries in the temporal bone, or in that met with in syphilitic disease of the bones, disease of the joints, and occasionally in tumors.

Multiple neuritis has a very complex etiology, the causes of which may be classified as follows: (*a*) The poisons of infectious diseases, as in leprosy, diphtheria, typhoid fever, small-pox, scarlet fever, and occasionally in other forms; (*b*) the organic poisons, comprising the diffusible stimulants, such as alcohol and ether, bisulphide of carbon and naphtha, and the metallic bodies, such as lead, arsenic, and mercury; (*c*) cachectic conditions, such as occur in anæmia, cancer, tuberculosis, or marasmus from any cause; (*d*) the endemic neuritis or beri-beri; and (*e*) lastly, there are cases in which none of these factors prevail, but the disease sets in suddenly after overexertion or exposure to cold.

Morbid Anatomy.—In neuritis due to the extension of inflammation the nerve is usually swollen, infiltrated, and red in color. The inflammation may be chiefly perineural or it may pass into the deeper portion—*interstitial* neuritis—in which form there is an accumulation of lymphoid elements between the nerve bundles. The nerve fibres themselves may not appear involved, but there is an increase in the nuclei of the sheath of Schwann. The myelin is fragmented, the nuclei of the internodal cells are swollen, and the axis-cylinders present varicosities or undergo granular degeneration. Ultimately the nerve fibres may be completely destroyed and replaced by a fibrous connective tissue in which much fat is sometimes deposited—the *lipomatous neuritis* of Leyden.

In other instances the condition is termed *parenchymatous* neuritis, in which the changes are like those met with in the secondary or Wallerian degeneration, which follows when the nerve fibre is cut off from the cell body of the neurone to which it belongs. The medullary substance and the axis-cylinders are chiefly involved, the interstitial tissue being but little altered or only affected secondarily. The muscles connected with the degenerated nerves usually show marked atrophic changes, and in some instances the change in the nerve sheath appears to extend directly to the interstitial tissue of the muscles—the *neuritis fascians* of Eichhorst.

Symptoms.—Localized Neuritis.—As a rule, the constitutional disturbances are slight. The most important symptom is pain of a boring or stabbing character, usually felt in the course of the nerve and in the parts to which it is distributed. The nerve itself is sensitive to pressure, probably, as Weir Mitchell suggests, owing to the irritation of its nervi nervorum. The skin may be slightly reddened or even œdematous over the seat of the inflammation. Mitchell has described increase in the temperature and sweating in the affected region, and such atrophic disturbances as effusion into the joints and herpes. The function of the muscle to which the nerve fibres are distributed is impaired, motion is painful, and there may be twitchings or contractions. The tactile sensation of the part may be somewhat deadened, even when the pain

is greatly increased. In the more chronic cases of local neuritis, such, for instance, as follow the dislocation of the humerus, the localized pain, which at first may be severe, gradually disappears, though some sensitiveness of the brachial plexus may persist for a long time, and the nerve cords may be felt to be swollen and firm. The pain is variable—sometimes intense and distressing; at others not causing much inconvenience. Numbness and formication may be present and the tactile sensation may be greatly impaired. The motor disturbances are marked. Ultimately there is extreme atrophy of the muscles. Contractures may occur in the fingers. The skin may be reddened or glossy, the subcutaneous tissue œdematous, and the nutrition of the nails may be defective. In the rheumatic neuritis subcutaneous fibroid nodules may develop.

A neuritis limited at first to a peripheral nerve may extend upward—the so-called ascending or migratory neuritis—and involve the larger nerve trunks, or even reach the spinal cord, causing subacute myelitis (Gowers). The condition is rarely seen in the neuritis from cold, or in that which follows fevers; but it occurs most frequently in traumatic neuritis.

J. K. Mitchell, in his monograph on injuries of nerves, concludes that the larger nerve trunks are most susceptible, and that the neuritis may spread either up or down, the former being the most common. The paralysis secondary to visceral disease, as of the bladder, may be due to an ascending neuritis. The inflammation may extend to the nerves of the other side, either through the spinal cord or its membranes, or without any involvement of the nerve-centres, the so-called sympathetic neuritis. The electrical changes in localized neuritis vary a great deal, depending upon the extent to which the nerve is injured. The lesion may be so slight that the nerve and the muscles to which it is distributed may react normally to both currents; or it may be so severe that the typical reaction of degeneration develops within a few days—i. e., the nerve does not respond to stimulation by either current, while the muscle reacts only to the galvanic current and in a peculiar manner. The contraction ~~caused~~ is slow and lazy, instead of sharp and quick as in the normal muscle, and the AC contraction is usually stronger than the KC contraction. Between these two extremes there are ~~many~~ different grades, and a careful electrical examination is most important ~~as an~~ aid ~~to~~ diagnosis and prognosis.

The duration varies from a few days to weeks or months. A slight traumatic neuritis may pass off in a day or two, while the severer cases, such as follow unreduced dislocation of the humerus may persist for months or never be completely relieved.

Multiple Neuritis.—The following are the most important groups ~~of~~ cases:

(*a*) *Acute Febrile Polyneuritis.*—The attack follows exposure to cold or overexertion, or, in some instances, comes on spontaneously. The onset resembles that of an acute infectious disease. There may be a definite chill, pains in the back and limbs or joints, so that the case may be thought to be rheumatic fever. The temperature rises rapidly and may reach 103° or 104° F. There are headache, loss of appetite, and the general symptoms of acute infection. The limbs and back ache. Intense pain in the nerves, however, is by no means constant. Tingling and formication are felt in the fingers and

toes, and there is increased sensitiveness of the nerve trunks or of the entire limb. Loss of muscular power, first marked, perhaps, in the legs, gradually comes on and extends with the features of an ascending paralysis. In other cases the paralysis begins in the arms. The extensors of the wrists and the flexors of the ankles are early affected, so that there is foot and wrist drop. In severe cases there is general loss of muscular power, producing a flabby paralysis, which may extend to the muscles of the face and to the intercostals, and respiration may be carried on by the diaphragm alone. The muscles soften and waste rapidly. There may be only hyperæsthesia with soreness and stiffness of the limbs; in some cases, increased sensitiveness with anæsthesia; in other instances the sensory disturbances are slight. The Argyll-Robertson pupil may be present and the pupils may be unequal. Involvement of the cranial nerves is rare, but the oculo-motor, the facial, and the fifth have been involved. The vagus may be attacked and the quickening of the pulse is usually attributed to this cause. Involvement of the bladder and rectum is rare, but it does occur in undoubted cases and does not necessarily mean involvement of the cord. The clinical picture is not to be distinguished, in many cases, from Landry's paralysis; in others, from the subacute myelitis of Duchenne.

The course is variable. In the most intense forms the patient may die in a week or ten days, with involvement of the respiratory muscles or from paralysis of the heart. As a rule, in cases of moderate severity, after persisting for five or six weeks, the condition remains stationary and then slow improvement begins. The paralysis in some muscles may persist for many months and contractures may occur from shortening of the muscles, but even when this occurs the outlook is, as a rule, good, although the paralysis may have lasted for a year or more.

(*b*) *Recurring Multiple Neuritis.*—Under the term *polyneuritis recurrens* Mary Sherwood has described from Eichhorst's clinic 2 cases in adults—in one case involving the nerves of the right arm, in the other both legs. In one patient there were three attacks, in the other two, the distribution in the various attacks being identical.

(*c*) *Alcoholic Neuritis.*—This, perhaps the most important form of multiple neuritis, was graphically described in 1822 by James Jackson, Sr., of Boston. Wilks recognized it as alcoholic paraplegia, but the starting point of the recent researches on the disease dates from the observations of Dumenil, of Rouen. It occurs most frequently in women, particularly in steady, quiet tipplers. Its appearance may be the first revelation to the physician or to the family of habits of secret drinking. The onset is usually gradual, and may be preceded for weeks or months by neuralgic pains and tingling in the feet and hands. Convulsions are not uncommon. Fever is rare. The paralysis gradually sets in, at first in the feet and legs, and then in the hands and forearms. The extensors are affected more than the flexors, so that there is wrist-drop and foot-drop. The paralysis may be thus limited and not extend higher in the limbs. In other instances there is paraplegia alone, while in the most extreme cases all the extremities are involved. In rare instances the facial muscles and the sphincters are also affected. The sensory symptoms are very variable. There are cases in which there are numbness and tingling only, without great pain. In other cases there are severe burning or boring pains,

the nerve trunks are sensitive, and the muscles are sore when grasped. The hands and feet are frequently swollen and congested, particularly when held down for a few moments. The cutaneous reflexes, as a rule, are preserved. The deep reflexes are usually lost.

The course of these alcoholic cases is, as a rule, favorable, and after persisting for weeks or months improvement gradually begins, the muscles regain their power, and even in the most desperate cases recovery may follow. The extensors of the feet may remain paralyzed for some time, and give to the patient a distinctive walk, the so-called *steppage* gait, characteristic of peripheral neuritis. It is sometimes known as the pseudo-tabetic gait, although in reality it could not well be mistaken for the gait of ataxia. The foot is thrown forcibly forward, the toe lifted high in the air so as not to trip upon it. The entire foot is slapped upon the ground as a flail. It is an awkward, clumsy gait, and gives the patient the appearance of constantly stepping over obstacles. Among the most striking features of alcoholic neuritis are the mental symptoms. Delirium is common, and there may be hallucinations with extravagant ideas, resembling somewhat those of general paralysis. In some cases the picture is that of ordinary delirium tremens, but the most peculiar and almost characteristic mental disorder is that so well described by Wilks, in which the patient loses all appreciation of time and place, and describes with circumstantial details long journeys which, he says, he has recently taken, or tells of persons whom he has just seen. This is the so-called Korsakoff's syndrome.

(*d*) *Multiple Neuritis in the Infectious Diseases.*—This has been already referred to, particularly in diphtheria, in which it is most common. The peripheral nature of the lesion in these instances has been shown by post mortem examination. The outlook is usually favorable and, except in diphtheria, fatal cases are uncommon. Multiple neuritis in tuberculosis, diabetes, and syphilis is of the same nature, being probably due to toxic materials absorbed into the blood.

(*e*) *The Metallic Poisons.*—Neuritis from arsenic may follow: (1) The medicinal use particularly of Fowler's solution. I have reported a case of Hodgkin's disease in which general neuritis was caused by ℥ j ♉ ij of the solution. In chorea a good many cases have been reported. Changes in the nails are not uncommon, chiefly the transverse ridging. In one case in my wards, of a young woman who had taken rough-on-rats, there were remarkable white lines—the leuconychia—running across the nails, without any special ridging. C. J. Aldrich finds that this is not uncommon in chronic arsenical poisoning. (2) The accidental contamination of food or drink. Chrome yellow may be used to color cakes, as in the cases recorded by D. D. Stewart. A remarkable epidemic of neuritis occurred in the Midland Counties of England, which was traced to the use of beer containing small quantities of arsenic, a contamination from the sulphuric acid used in making glucose. Reynolds, who studied these cases, believes that most of the instances of neuritis in drinkers are arsenical, but admits that the slight cases may be due to the alcohol itself. Pigmentation of the skin is an important distinguishing sign. The general features have been referred to under arsenical poisoning. Lead is a much more frequent cause. Neuritis has followed the use of mercurial inunctions. Zinc is a rare cause; I saw a case with Urban

Smith which followed the use of two grains of the sulpho-carbolate taken daily for three years. Tea, coffee, and tobacco are mentioned as rare causes.

(*f*) *Endemic neuritis, beri-beri,* has been considered elsewhere.

Anæsthesia Paralysis.—Here perhaps may most appropriately be considered the forms of paralysis following the use of anæsthetics, or of too long-continued compression during operations. Much has been written in the past few years upon this subject. There are two groups of cases:

(*a*) During an operation the nerves may be compressed, either the brachial plexus by the humerus or the musculo-spiral by the table. The pressure most frequently occurs when the arm is elevated alongside the head, as in laparotomy done in the Trendelenburg position, or held out from the body, as in breast amputations. Instances of paralysis of the crural nerves by leg-holders are also reported. The too firm application of a tourniquet may be followed by a severe paralysis.

(*b*) Paralysis from cerebral lesions during etherization. In one of Garrigues' cases paralysis followed the operation, and at the autopsy, seven weeks later, softening of the brain was found. Apoplexy or embolism may occur during anæsthesia. In Montreal a cataract operation was performed on an old man. He did not recover from the anæsthetic; I found post mortem a cerebral hæmorrhage. A man was admitted to the Philadelphia Hospital, completely comatose, who on the previous day had been given ether for a minor operation. He never recovered consciousness, but remained deeply comatose, with great muscular relaxation, low temperature, 97.5°, and noisy respirations; he died two days later. There was, unfortunately, no autopsy. Epileptic convulsions may occur during the anæsthesia, and may even prove fatal. The possibility has to be considered of paralysis from loss of blood in prolonged operations, though I have no personal knowledge of any such cases.

And, lastly, a paralysis might result from the toxic effects of the ether in a very protracted administration.

Diagnosis.—The electrical condition in multiple neuritis is thus described by Allen Starr: "The excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost and then the muscles contract to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and thus far it is quite pathognomonic of neuritis. For in anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion.

"The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found and the negative pole produces stronger contractions than the positive pole. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle and nerve are therefore important symptoms of multiple neuritis."

There is rarely any difficulty in distinguishing the alcohol cases. The

combination of wrist and foot drop with congestion of the hands and feet, and the peculiar delirium already referred to, are quite characteristic. The rapidly advancing cases with paralysis of all extremities, often reaching to the face and involving the sphincters, are more commonly regarded as of spinal origin, but the general opinion seems to point strongly to the fact that all such cases are peripheral. The less acute cases, in which the paralysis gradually involves the legs and arms with rapid wasting, simulate closely and are usually confounded with the subacute atrophic spinal paralysis of Duchenne. The diagnosis from locomotor ataxia is rarely difficult. The *steppage* gait is entirely different from that of tabes. There is rarely positive incoördination. The patient can usually stand well with the eyes closed. Foot-drop is not common in locomotor ataxia. The lightning pains are absent and there are usually no pupillary symptoms. The etiology, too, is of moment. The patient is recovering from a paralysis which has been more extensive, or from arsenical poisoning, or he has diabetes.

Treatment.—Rest in bed is essential. In the acute cases with fever the salicylates and antipyrin are recommended. To allay the intense pain morphia or the hot applications of lead water and laudanum are often required. Great care must be exercised in treating the alcoholic form, and the physician must not allow himself to be deceived by the statements of the relatives. It is sometimes exceedingly difficult to get a history of spirit drinking. In the alcoholic form it is well to reduce the stimulants gradually. If there is any tendency to bed-sores an air-bed should be used or the patient placed in a continuous bath. Gentle friction of the muscles may be applied from the outset, and in the later stages, when the atrophy is marked and the pains have lessened, massage is probably the most reliable means at our command. Contractures may be gradually overcome by passive movements and extension. Often with the most extreme deformity from contracture, recovery is, in time, still possible. The interrupted current is useful when the acute stage is passed.

Of internal remedies, strychnia is of value and may be given in increasing doses. Arsenic also may be employed, and if there is a history of syphilis the iodide of potassium and mercury may be given.

II. NEUROMATA

Tumors situated on nerve fibres may consist of nerve substance proper, the true neuromata, or of fibrous tissue, the false neuromata. The true neuroma usually contains nerve fibres only, or in rare instances ganglion cells. Cases of ganglionic or medullary neuroma are extremely rare; some of them, as Lancereaux suggests, are undoubtedly instances of malformation of the brain substance. In other instances the tumor is, in all probability, a glioma with cells closely resembling those of the central nervous system. The growths are often intermediate in their anatomical structure between the true and the false.

Plexiform Neuroma.—In this remarkable condition the various nerve cords may be occupied by many hundreds of tumors. The cases are often hereditary and usually congenital. The tumors may occur in all the nerves of the body,

Painful menses: rebellious to other [illegible] (causalgia). treated by injection of alcohol into nerve trunk directly. (60–80% alcohol) Shaved before injected area.

and, as numbers of them may be made out on palpation, the diagnosis is usually easy. One of the most remarkable cases is that described by Prudden, the specimens of which are in the medical museum of Columbia College, New York. There were over 1,182 distinct tumors distributed on the nerves of the body. These tumors rarely are painful, but may cause symptoms through pressure on neighboring structures.

Generalized Neuro-fibromatosis: von Recklinghausen's Disease.—Special attention was first directed to this particular form of multiple neuroma by von Recklinghausen in 1882. The disease presents four essential features:

(*a*) Soft, fibrous nodules, some sessile, others pedunculated, varying greatly in size and number, are scattered over the surface of the body. These subcutaneous growths at times may be diffuse and reach an enormous size, producing a condition called "Elephantiasis Neuromatosa."

(*b*) Tumors resembling those of plexiform neuroma may be present on any part of the nerve trunks from their central origin to the periphery. Their variable situation may lead to a variety of symptoms, more especially as they may arise from the nerve roots within the spinal canal or cranium. Superficial painful nodules may also be present.

(*c*) Patches of brownish pigmentation of the skin, either as small spots or large areas, are always present. Congenital nævi are a frequent accompaniment of the disease.

(*d*) There are many variable sensory or motor phenomena resulting from the presence of the nerve tumors, but peculiar mental changes, with loss of intellectual power and sometimes difficulty in speaking, are especially characteristic of the disease.

Three generations have been affected. A sarcomatous change has been present in some tumors, and in a few cases associated brain tumors, as gliomata, have been present. The tumors are believed to originate in the sheath of Schwann, in confirmation of which is the interesting point that the optic and olfactory nerves which are devoid of this sheath have never been found affected with neuromatosis.

The prognosis depends on the possibility of successful removal of such tumors as are causing greatest inconvenience.

"Tubercula Dolorosa."—Multiple neuromata may especially affect the terminal cutaneous branches of the sensory nerves and lead to small subcutaneous painful nodules, often found on the face, breast, or about the joints. They may be associated with tumors of the nerve trunks.

"Amputation Neuromata."—These bulbous swellings may form on the central ends of nerves which have been divided in injuries or operations. They are especially common after amputations. They are due to the tangled coil of axis-cylinder processes growing down from the central stump in an effort to reach their former end structures. They are very painful and usually require surgical removal, but often recur.

III. DISEASES OF THE CEREBRAL NERVES

OLFACTORY NERVES AND TRACTS

The functions of the olfactory nerves may be disturbed at their origin, in the nasal mucous membrane, at the bulb, in the course of the tract, or at the centres in the brain. The disturbances may be manifested in subjective sensations of smell, complete loss of the sense, and occasionally in hyperæsthesia.

Subjective Sensations; Parosmia.—Hallucinations of this kind are found in the insane and in epilepsy. The aura may be represented by an unpleasant odor, described as resembling chloride of lime, burning rags, or feathers. In a few cases with these subjective sensations tumors have been found in the hippocampi. In rare instances, after injury of the head, the sense is perverted—odors of the most different character may be alike, or the odor may be changed, as in a patient noted by Morell Mackenzie, who for some time could not touch cooked meat, as it smelt to her exactly like stinking fish.

Increased sensitiveness (hyperosmia) occurs chiefly in nervous, hysterical women, in whom it may sometimes be developed so greatly that, like a dog, they can recognize the difference between individuals by the odor alone.

Anosmia; Loss of the Sense of Smell.—This may be produced by: (*a*) Affections of the origin of the nerves in the mucous membrane, which is perhaps the most frequent cause. It is by no means uncommon in association with chronic nasal catarrh and polypi. In paralysis of the fifth nerve, the sense of smell may be lost on the affected side, owing to interference with the secretion.

It is doubtful whether the cases of loss of smell following the inhalations of very foul or strong odors should come under this or under the central division.

(*b*) Lesions of the bulbs or of the tracts. In falls or blows, in caries of the bones, and in meningitis or tumor, the bulbs or the olfactory tracts may be involved. After an injury to the head the loss of smell may be the only symptom. Mackenzie notes a case of a surgeon who was thrown from his gig and lighted on his head. The injury was slight, but the anosmia which followed was persistent. In locomotor ataxia the sense of smell may be lost, possibly owing to atrophy of the nerves.

(*c*) Lesions of the olfactory centres. There are congenital cases in which the structures have not been developed. Cases have been reported by Beevor, Hughlings Jackson, and others, in which anosmia has been associated with disease in the hemisphere.

To test the sense of smell the pungent bodies, such as ammonia, which act upon the fifth nerve, should not be used, but such substances as cloves, peppermint, and musk. This sense is readily tested as a routine matter in brain cases by having two or three bottles containing the essential oils. In all instances a rhinoscopic examination should be made, as the condition may be due to local, not central causes. The *treatment* is unsatisfactory even in the cases due to local lesions in the nostrils.

OPTIC NERVE AND TRACT

(1) *Lesions of the Retina*

These are of importance to the physician, and information of the greatest value may be obtained by a systematic examination of the eye grounds. Only a brief reference can here be made to the more important of the appearances.

Retinitis.—This occurs in certain general affections, more particularly in Bright's disease, syphilis, leukæmia, and anæmia. The common feature in all these states is the occurrence of hæmorrhage and the development of opacities. There may also be a diffuse cloudiness due to effusion of serum. The hæmorrhages are in the layer of nerve fibres. They vary greatly in size and form, but often follow the course of vessels. When recent the color is bright red, but they gradually change and old hæmorrhages are almost black. The white spots are due either to fibrinous exudate or to fatty degeneration of the retinal elements, and occasionally to accumulation of leucocytes or to a localized sclerosis of the retinal elements. The more important of the forms of retinitis to be recognized are:

Albuminuric retinitis, which occurs in chronic nephritis, particularly in the interstitial or contracted form. The percentage of cases affected is from 15 to 25. There are instances in which these retinal changes are associated with the granular kidney at a stage when the amount of albumin may be slight or transient; but in all such instances it will be found that there is a marked arterio-sclerosis. Gowers recognizes a degenerative form (most common), in which, with the retinal changes, there may be scarcely any alteration in the disk; a hæmorrhagic form, with many hæmorrhages and but slight signs of inflammation; and an inflammatory form, in which there is much swelling of the retina and obscuration of the disk. It is noteworthy that in some instances the inflammation of the optic nerve predominates over the retinal changes, and one may be in doubt for a time whether the condition is really associated with the renal changes or dependent upon intracranial disease.

Syphilitic Retinitis.—In the acquired form this is less common than choroiditis. In inherited syphilis *retinitis pigmentosa* is sometimes met with.

Retinitis in Anæmia.—It has long been known that a patient may become blind after a large hæmorrhage, either suddenly or within two or three days, and in one or both eyes. Occasionally the loss may be permanent and complete. In some of these instances a neuro-retinitis has been found, probably sufficient to account for the symptoms. In the more chronic anæmias, particularly in the pernicious form, retinitis is common, as determined first by Quincke.

In malaria retinitis or neuro-retinitis may be present, as noted by Stephen Mackenzie. It is seen only in the chronic cases with anæmia, and in my experience is not nearly so common proportionately as in pernicious anæmia.

Leukæmic Retinitis.—In this affection the retinal veins are large and distended; there is also a peculiar retinitis, as described by Liebreich. It is not very common. There are numerous hæmorrhages and white or yellow areas, which may be large and prominent. In one of my cases the retina post

mortem was dotted with many small, opaque, white spots, looking like little tumors, the larger of which had a diameter of nearly 2 mm.

Retinitis is also found occasionally in diabetes, in purpura, in chronic lead poisoning, and sometimes as an idiopathic affection.

Functional Disturbances of Vision.—(*a*) Toxic Amaurosis.—This occurs in uræmia and may follow convulsions or come on independently. The condition, as a rule, persists only for a day or two. This form of amaurosis occurs in poisoning by lead, alcohol, and occasionally by quinine. It seems more probable that the poisons act on the centres and not on the retina.

(*b*) Tobacco Amblyopia.—The loss of sight is usually gradual, equal in both eyes, and affects particularly the centre of the field of vision. The eye-grounds may be normal, but occasionally there is congestion of the disks. On testing the color fields a central scotoma for red and green is found in all cases. Ultimately, if the use of tobacco is continued, organic changes may develop with atrophy of the disk.

(*c*) Hysterical Amaurosis.—More frequently this is loss of acuteness of vision—amblyopia—but the loss of sight in one or both eyes may apparently be complete. The condition will be mentioned subsequently under hysteria.

(*d*) Night-blindness—nyctalopia—the condition in which objects are clearly seen during the day or by strong artificial light, but become invisible in the shade or in twilight, and *hemeralopia*, in which objects can not be clearly seen without distress in daylight or in a strong artificial light, but are readily seen in a deep shade or in twilight, are functional anomalies of vision, which rarely come under the notice of the physician. It may occur in epidemic form.

(*e*) Retinal hyperæsthesia is sometimes seen in hysterical women, but is not found frequently in actual retinitis. I have seen it once, however, in albuminuric retinitis, and once, in a marked degree, in a patient with aortic insufficiency, in whose retinæ there were no signs other than the throbbing arteries.

(2) *Lesions of the Optic Nerve*

Optic Neuritis (*Papillitis; Choked Disk*).—In the first stage there is congestion of the disk and the edges are blurred and striated. In the second stage the congestion is more marked; the swelling increases, the striation also is more visible. The physiological cupping disappears and hæmorrhages are not uncommon. The arteries present little change, the veins are dilated, and the disk may swell greatly. In slight grades of inflammation the swelling gradually subsides and occasionally the nerve recovers completely. In instances in which the swelling and exudate are very great the subsidence is slow, and when it finally disappears there is complete atrophy of the nerve. The retina not infrequently participates in the inflammation, which is then a neuro-retinitis.

This condition is of the greatest importance in diagnosis. It may exist in its early stages without any disturbance of vision, and even with extensive papillitis the sight may for a time be good.

Optic neuritis is seen occasionally in anæmia and lead poisoning, more commonly in Bright's disease as neuro-retinitis. It occurs occasionally as a primary idiopathic affection. The frequent connection with intracranial

disease, particularly tumor, makes its presence of great value to practitioners. The nature of the growth is without influence. In over 90 per cent. of such instances the papillitis is bilateral. It is also found in meningitis, either the tuberculous or the simple form. In meningitis it is easy to see how the inflammation may extend down the nerve sheath. In the case of tumor, however, it is probable that mechanical conditions, especially the venous stasis, are alone responsible for the œdematous swelling. It often subsides very rapidly after a palliative craniectomy has been performed.

Optic Atrophy.—This may be: (*a*) A primary affection. There is an hereditary form, in which the disease has developed in all the males of a family shortly after puberty. A large number of the cases of primary atrophy are associated with spinal disease, particularly locomotor ataxia. Other causes which have been assigned for the primary atrophy are cold, sexual excesses, diabetes, the specific fevers, methyl alcohol, and lead.

(*b*) Secondary atrophy results from cerebral diseases, pressure on the chiasma or on the nerves, or, most commonly of all, as a sequence of papillitis.

The ophthalmoscopic appearances are different in the cases of primary and secondary atrophy. In the former the disk has a gray tint, the edges are well defined, and the arteries look almost normal; whereas in the consecutive atrophy the disk has a staring opaque white aspect, with irregular outlines, and the arteries are very small.

The symptom of optic atrophy is loss of sight, proportionate to the damage in the nerve. The change is in three directions: "(1) Diminished acuity of vision; (2) alteration in the field of vision; and (3) altered perception of color" (Gowers). The outlook in primary atrophy is bad.

(3) *Affections of the Chiasma and Tract.*

At the chiasma the optic nerves undergo partial decussation. Each optic tract, as it leaves the chiasma, contains nerve fibres which originate in the retinæ of both eyes. Thus, of the fibres of the right tract, part have come through the chiasma without decussating from the temporal half of the right retina, the other and larger portion of the fibres of the tract have decussated in the chiasma, coming as they do from the left optic nerve and the nasal half of the retina on the left side. The fibres which cross are in the middle portion of the chiasma, while the direct fibres are on each side. The following are the most important changes which ensue in lesions of the tract and of the chiasma:

Unilateral Affection of Tract.—If on the right side, this produces loss of function in the temporal half of the retina on the right side, and in the nasal half of the retina on the left side, so that there is only half vision, and the patient is blind to objects on the left side. This is termed homonymous hemianopia or lateral hemianopia. The fibres passing to the right half of each retina being involved, the patient is blind to objects in the left half of each visual field. The hemianopia may be partial and only a portion of the half field may be lost. The unaffected visual fields may have the normal extent, but in some instances there is considerable reduction. When the left half of one field and the right half of the other, or *vice versa* are blind, the condition is known as heteronymous hemianopia.

Disease of the Chiasma.—(*a*) A lesion involves, as a rule, chiefly the central portion, in which the decussating fibres pass which supply the inner or nasal halves of the retinæ, producing in consequence loss of vision in the outer half of each field, or what is known as temporal hemianopia.

(*b*) If the lesion is more extensive it may involve not only the central portion, but also the direct fibres on one side of the commissure, in which case there would be total blindness in one eye and temporal hemianopia in the other.

(*c*) Still more extensive disease is not infrequent from pressure of tumors in this region, the whole chiasma is involved, and total blindness results. The different stages in the process may often be traced in a single case from temporal hemianopia, then complete blindness in one eye with temporal hemianopia in the other, and finally complete blindness.

(*d*) A limited lesion of the outer part of the chiasma involves only the direct fibres passing to the temporal halves of the retinæ and inducing blindness in the nasal field, or, as it is called, nasal hemianopia. This, of course, is extremely rare. Double nasal hemianopia may occur as a manifestation of tabes and in tumors involving the outer fibres of each tract.

(4) *Affections of the Tract and Centres*

The optic tract crosses the crus (cerebral peduncle) to the hinder part of the optic thalamus and divides into two portions, one of which (the lateral root) goes to the pulvinar of the thalamus, the lateral geniculate body, and to the anterior quadrigeminal body (superior colliculus). From these parts, in which the lateral root terminates, fibres pass into the posterior part of the internal capsule and enter the occipital lobe, forming the fibres of the optic radiation, which terminate in and about the cuneus, the region of the visual perceptive centre. The fibres of the medial division of the tract pass to the medial geniculate body and to the posterior quadrigeminal body. The medial root contains the fibres of the commissura inferior of v. Gudden, which are believed to have no connection with the retinæ. It is still held by some physiologists that the cortical visual centre is not confined to the occipital lobe alone, but embraces the occipito-angular region.

A lesion of the fibres of the optic path anywhere between the cortical centre and the chiasma will produce hemianopia. The lesion may be situated: (*a*) In the optic tract itself. (*b*) In the region of the thalamus, lateral geniculate body, and the corpora quadrigemina, into which the larger part of each tract enters. (*c*) A lesion of the fibres passing from the centres just mentioned to the occipital lobe. This may be either in the hinder part of the internal capsule or the white fibres of the optic radiation. (*d*) Lesion of the cuneus. Bilateral disease of the cuneus may result in total blindness. (*e*) There is clinical evidence to show that lesion of the angular gyrus may be associated with visual defect, not so often hemianopia as crossed amblyopia, dimness of vision in the opposite eye, and great contraction in the field of vision. Lesions in this region are associated with mind-blindness, a condition in which there is failure to recognize the nature of objects.

The effects of lesions in the optic nerve in different situations from the retinal expansion to the brain cortex are as follows: (1) Of the optic nerve—total

blindness of the corresponding eye; (2) of the optic chiasma, either temporal hemianopia, if the central part alone is involved, or nasal hemianopia, if the lateral region of each chiasma is involved; (3) lesion of the optic tract between the chiasma and the lateral geniculate body produces lateral hemianopia; (4) lesion of the central fibres of the nerve between the geniculate bodies and the cerebral cortex produces lateral hemianopia; (5) lesion of the cuneus causes lateral hemianopia; and (6) lesion of the angular gyrus may be associated with hemianopia, sometimes crossed amblyopia, and the condition known as mind-blindness. (See Fig. 19, with accompanying explanation.)

Diagnosis of Lesions of the Optic Nerve and Tract.—Having determined the presence of hemianopia, the question arises as to the situation of the lesion, whether in the tract between the chiasma and the geniculate bodies or in the central portion of the fibres between these bodies and the visual centres. This can be determined in some cases by the test known as Wer-

not perceived. Hemianæsthesia is not uncommon in such cases, owing to the close association of the sensory and visual tracts at the posterior part of the internal capsule. Certain forms of aphasia also occur in many of the cases.

The optic aphasia of Freund may be mentioned here. The patient, after an apoplectic attack, though able to recognize ordinary objects shown to him,

is unable to name them correctly. If he be permitted to touch the object he may be able to name it quickly and correctly. Freund's optic aphasia differs from mind-blindness, since in the latter affection the objects seen are not recognized. Optic aphasia, like word-blindness, never occurs alone, but is always associated with hemianopia, or mind-blindness, and often also with word-deafness. In the cases which have thus far come to autopsy there has always been a lesion in the white matter of the occipital lobe on the left side.

MOTOR NERVES OF THE EYEBALL

Third Nerve (*Nervus oculomotorius*).—The nucleus of origin of this nerve is situated in the floor of the aqueduct of Sylvius; the nerve passes through the crus at the side of which it emerges. Passing along the wall of the cavernous sinus, it enters the orbit through the sphenoidal fissure and supplies, by its superior branch, the levator palpebræ superioris and the superior rectus, and by its inferior branch the internal and inferior recti muscles and the inferior oblique. Branches pass to the ciliary muscle and the constrictor of the iris. Lesions may affect the nucleus or the nerve in its course and cause either paralysis or spasm.

Paralysis.—A nuclear lesion is usually associated with disease of the centres for the other eye muscles, producing a condition of general ophthalmoplegia. More commonly the nerve itself is involved in its course, either by meningitis, gummata, or aneurism, or is attacked by a neuritis, as in diphtheria and locomotor ataxia. Complete paralysis of the third nerve is accompanied by the following symptoms:

Paralysis of all the muscles, except the superior oblique and external rectus, by which the eye can be moved outward and a little downward and inward. There is divergent strabismus. There is ptosis or drooping of the upper eyelid, owing to paralysis of the levator palpebræ. The pupil is usually dilated. It does not contract to light, and the power of accommodation is lost. The most striking features of this paralysis are the external strabismus, with diplopia or double vision, and the ptosis. In very many cases the affection of the third nerve is partial. Thus the levator palpebræ and the superior rectus may be involved together, or the ciliary muscles and the iris may be affected and the external muscles may escape.

There is a remarkable form of recurring oculo-motor paralysis affecting chiefly women, and involving all the branches of the nerve. In some cases the attacks have come on at intervals of a month; in others a much longer period has elapsed. The attacks may persist throughout life. They are sometimes associated with pain in the head and sometimes with migraine. Mary Sherwood has collected from the literature 23 cases.

Ptosis is a common and important symptom in nervous affections. We may here briefly refer to the conditions under which it may occur: (*a*) A congenital, incurable form, which is frequently seen; (*b*) the form associated with definite lesion of the third nerve, either in its course or at its nucleus. This may come on with paralysis of the superior rectus alone or with paralysis of the internal and inferior recti as well. (*c*) There are instances of complete or partial ptosis associated with cerebral lesions without any other branch of the third nerve being paralyzed. The exact position of the cortical centre

or centres is as yet unknown. (*d*) Hysterical ptosis, which is double and occurs with other hysterical symptoms. (*e*) Pseudo-ptosis, due to affection of the sympathetic nerve, is associated with symptoms of vaso-motor palsy, such as elevation of the temperature on the affected side with redness and œdema of the skin. Contraction of the pupil exists on the same side and the eyeball appears rather to have shrunk into the orbit. (*f*) In idiopathic muscular atrophy, when the face muscles are involved, there may be marked bilateral ptosis. And, lastly, in weak, delicate women there is often to be seen a transient ptosis, particularly in the morning.

Among the most important of the symptoms of the third-nerve paralysis are those which relate to the ciliary muscle and iris.

Cyclоplegia, paralysis of the ciliary muscle, causes loss of the power of accommodation. Distant vision is clear, but near objects can not be properly seen. In consequence the vision is indistinct, but can be restored by the use of convex glasses. This may occur in one or in both eyes; in the latter case it is usually associated with disease in the nuclei of the nerve. Cycloplegia is an early and frequent symptom in diphtheritic paralysis and occurs also in tabes.

Iridoplegia, or paralysis of the iris, occurs in three forms (Gowers):

(*a*) *Accommodation iridoplegia,* in which the pupil does not diminish in size during the act of accommodation. To test for this the patient should look first at a distant and then at a near object in the same line of vision.

(*b*) *Reflex Iridoplegia.*—The path for the iris reflex is along the optic nerve and tract to its termination, then to the nucleus of the third nerve, and along the trunk of this nerve to the ciliary ganglion, and so through the ciliary nerves to the eyes. Each eye should be tested separately, the other one being covered. The patient should look at a distant object in a dark part of the room; then a light is brought suddenly in front of the eye at a distance of three or four feet, so as to avoid the effect of accommodation. Loss of this iris reflex with retention of the accommodation contraction is known as the Argyll-Robertson pupil.

(*c*) *Loss of the Skin Reflex.*—If the skin of the neck is pinched or pricked the pupil dilates reflexly, the afferent impulses being conveyed along the cervical sympathetic. Erb pointed out that this skin reflex is lost usually in association with the reflex contraction, but the two are not necessarily conjoined. In iridoplegia the pupils are often small, particularly in spinal disease, as in the characteristic small pupils of tabes—spinal myosis. Iridoplegia may coexist with a pupil of medium size.

Inequality of the pupils—anisocoria—is not infrequent in progressive paresis and in tabes. It may also occur in perfectly healthy individuals.

Spasm.—Occasionally in meningitis and in hysteria there is spasm of the muscles supplied by the third nerve, particularly the internal rectus and the levator palpebræ. The clonic rhythmical spasm of the eye muscles is known as *nystagmus*, in which there is usually a bilateral, rhythmical, involuntary movement of the eyeballs. The condition is met with in many congenital and acquired brain lesions, in albinism, and sometimes in coal miners.

Fourth Nerve (*Nervus trochlearis*).—This supplies the superior oblique muscle. In its course around the outer surface of the crus and in its pas-

sage into the orbit it is liable to be compressed by tumors, by aneurism, or in the exudation of basilar meningitis. Its nucleus in the upper part of the fourth ventricle may be involved by tumors or undergo degeneration with the other ocular nuclei. The superior oblique muscle acts in such a way as to direct the eyeball downward and rotate it slightly. The paralysis causes defective downward and inward movement, often too slight to be noticed. The head is inclined somewhat forward and toward the sound side, and there is double vision when the patient looks down.

Sixth Nerve (*Nervus abducens*).—This nerve emerges at the junction of the pons and medulla, then, passing forward, it enters the orbit and supplies the external rectus muscle. Owing to its long course and exposed position it is more commonly injured than any other cranial nerve. It is affected by meningitis at the base, by gummata or other tumors, and sometimes by cold. There is internal strabismus, and the eye can not be turned outward. Diplopia occurs on looking toward the paralyzed side.

"When the nucleus is affected there is, in addition to paralysis of the external rectus, inability of the internal rectus of the opposite eye to turn that eye inward. As a consequence of this the axes of the eyes are kept parallel, and both are conjugately deviated to the opposite side, away from the side of lesion. The reason of this is that the nucleus of the sixth nerve sends fibres up in the pons to that part of the nucleus of the opposite third nerve which supplies the internal rectus. We thus have paralysis of the internal rectus without the nucleus of the third nerve being involved, owing to its receiving its nervous impulses for parallel movement from the sixth nucleus of the opposite side. As the sixth nucleus is in such proximity to the facial nerve in the substance of the pons, it is frequently found that the whole of the face on the same side is paralyzed, and gives the electrical reaction of degeneration, so that with a lesion of the *left* sixth nucleus there is conjugate deviation of both eyes to the *right*—i. e., paralysis of the left external and the right internal rectus, and sometimes complete paralysis of the *left* side of the face" (Beevor).

General Features of Paralysis of the Motor Nerves of the Eye.—Gowers divides them into five groups:

(*a*) *Limitation of Movement.*—Thus, in paralysis of the external rectus, the eyeball can not be moved outward. When the paralysis is incomplete the movement is deficient in proportion to the degree of the palsy.

(*b*) *Strabismus.*—The axes of the eyes do not correspond. Thus, paralysis of the internal rectus causes a divergent squint; of the external rectus, a convergent squint. At first this is evident only when the eyes are moved in the direction of the action of the weak muscle, but may become constant by the contraction of the opposing muscle. The deviation of the axis of the affected eye from parallelism with the other is called the primary deviation.

(*c*) *Secondary Deviation.*—If, while the patient is looking at an object, the sound eye is covered, so that he fixes the object looked at with the affected eye only, the sound eye is moved still further in the same direction—e. g., outward, when there is paralysis of the opposite internal rectus. This is known as secondary deviation. It depends upon the fact that, if two muscles are acting together, when one is weak and an effort is made to contract it, the

increased effort—innervation—acts powerfully upon the other muscle, causing an increased contraction.

(*d*) *Erroneous Projection.*—"We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles" (Gowers). With the eyes at rest in the mid-position, an object at which we are looking is directly opposite our face. Turning the eyes to one side, we recognize that object in the middle of the field or to the side of this former position. We estimate the degree by the amount of movement of the eyes, and when the object moves and we follow it we judge of its position by the amount of movement of the eyeballs. When one ocular muscle is weak the increased innervation gives the impression of a greater movement of the eye than has really taken place. The mind, at the same time, receives the idea that the object is further on one side than it really is, and in an attempt to touch it the finger may go beyond it. As the equilibrium of the body is in a large part maintained by a knowledge of the relation of external objects to it obtained by the action of the eye muscles, this erroneous projection resulting from paralysis disturbs the harmony of these visual impressions and may lead to giddiness—ocular vertigo.

(*e*) *Double Vision.*—This is one of the most disturbing features of paralysis of the eye muscles. The visual axes do not correspond, so that there is a double image—diplopia. That seen by the sound eye is termed the true image; that by the paralyzed eye, the false. In simple or homonymous diplopia the false image is "on the same side of the other as the eye by which it is seen." In crossed diplopia it is on the other side. In convergent squint the diplopia is simple; in divergent it is crossed.

Ophthalmoplegia.—Under this term is described a chronic progressive paralysis of the ocular muscles. Two forms are recognized—ophthalmoplegia *externa* and ophthalmoplegia *interna*. The conditions may occur separately or together and are described by Gowers under nuclear ocular palsy.

Ophthalmoplegia externa.—The condition is one of more or less complete palsy of the external muscles of the eyeball, due usually to a slow degeneration in the nuclei of the nerves, but sometimes to pressure of tumors or to basilar meningitis. It is often, but not necessarily, associated with ophthalmoplegia interna. Of 62 cases analyzed by Siemerling in only 11 could syphilis be positively determined. The levator muscles of the eyelids and the superior recti are first involved, and gradually the other muscles, so that the eyeballs are fixed and the eyelids droop. There is sometimes slight protrusion of the eyeballs. The disease is essentially chronic and may last for many years. It is found particularly in association with general paralysis, ~~locomotor ataxia~~, and in progressive muscular atrophy. Mental disorders were present in 11 of the 62 cases. With it may be associated atrophy of the optic nerve and affections of other cerebral nerves. Occasionally, ~~as noted by~~ (Bristowe), it may be functional.

Ophthalmoplegia interna.—Jonathan Hutchinson applied this term to a progressive paralysis of the internal ocular muscles, causing loss of pupillary action and the power of accommodation. When the internal and external muscles are involved the affection is known as total ophthalmoplegia,

and in a majority of the cases the two conditions are associated. In some instances the internal form may depend upon disease of the ciliary ganglion.

While, as a rule, ophthalmoplegia is a chronic process, there is an acute form associated with hæmorrhagic softening of the nuclei of the ocular muscles. There is usually marked cerebral disturbance. It was to this form that Wernicke gave the name poliencephalitis superior.

Treatment of Ocular Palsies.—It is important to ascertain, if possible, the cause. The forms associated with locomotor ataxia are obstinate, and resist treatment. Occasionally, however, a palsy, complete or partial, may pass away spontaneously. The group of cases associated with chronic degenerative changes, as in progressive paresis and bulbar paralysis, is little affected by treatment. On the other hand, in syphilitic cases, mercury and iodide of potassium ~~are indicated and~~ are often beneficial. Arsenic and strychnia, the latter hypodermically, may be employed. In any case in which the onset is acute, with pain, hot fomentations and counter-irritation ~~or leeches~~ applied to the temple give relief. The direct treatment by electricity has been extensively employed, but probably without any special effect. The diplopia may be relieved by the use of prisms, or it may be necessary to cover the affected eye ~~with an opaque glass~~.

FIFTH NERVE

(*Nervus trigeminus*)

Etiology.—Paralysis may result from: (*a*) Disease of the pons, particularly hæmorrhage or patches of sclerosis. (*b*) Injury or disease at the base of the brain. Fracture rarely involves the nerve; on the other hand, meningitis, acute or chronic, and caries of the bone are not uncommon causes. (*c*) The branches may be affected as they pass out—the first division by tumors pressing on the cavernous sinus or by aneurism; the second and third divisions by growths which invade the spheno-maxillary fossa. (*d*) Primary neuritis, which is rare.

Symptoms.—(*a*) Sensory Portion.—Disease of the fifth nerve may cause loss of sensation in the parts supplied, including the half of the face, the corresponding side of the head, the conjunctiva, the mucosa of the lips, tongue, hard and soft palate, and of the nose of the same side. The anæsthesia may be preceded by tingling or pain. The muscles of the face are also insensible and the movements may be slower. The sense of smell is interfered with, owing to dryness of the mucous membrane. There may be disturbance of the sense of taste. The salivary, lachrymal, and buccal secretions may be lessened, and the teeth may become loose. Unless properly guarded from injury an ulcerative inflammation of the eye may follow. This was ~~formerly~~ supposed to be due to nutritional changes from paralysis of so-called trophic nerve fibres. This idea has ~~of late years~~ been overthrown by the large number of cases in which the Gasserian ganglion has been removed ~~for obstinate neuralgia~~ without consequent inflammation of the eye. Herpes may occur in the region supplied by the nerve, usually the upper branch, and is associated with much pain, which may be peculiarly enduring, lasting for months or years (Gowers). In herpes zoster with the neuritis there may be

slight enlargement of the cervical glands. (See under Neuralgia for Tic Douloureux.)

(*b*) Motor Portion.—The inability to use the muscles of mastication on the affected side is the distinguishing feature of paralysis of this portion of the nerve. It is recognized by placing the finger on the masseter and temporal muscles, and, when the patient closes the jaw, the feebleness of their contraction is noted. If paralyzed, the external pterygoid can not move the jaw toward the unaffected side; and when depressed, the jaw deviates to the paralyzed side. The motor paralysis of the fifth nerve is almost invariably a result of involvement of the nerve after it has left the nucleus. Cases, however, have been associated with cortical lesions. The cortical motor centre for the trigeminus, or for movements effecting closure of the jaw, lies below that for movements of the face at the lower part of the anterior central convolution.

Spasm of the Muscles of Mastication.—Trismus, the masticatory spasm of Romberg, may be tonic or clonic, and is either an associated phenomenon in general convulsions or, more rarely, an independent affection. In the tonic form the jaws are kept close together—lock-jaw—or can be separated only for a short space. The muscles of mastication can be seen in contraction and felt to be hard; the spasm is often painful. This tonic contraction is an early symptom in tetanus, and is sometimes seen in tetany. A form of this tonic spasm occurs in hysteria. Occasionally trismus follows exposure to cold, and is said to be due to reflex irritation from the teeth, the mouth, or caries of the jaw. It may also be a symptom of organic disease due to irritation near the motor nucleus of the fifth nerve.

Clonic spasm of the muscles supplied by the fifth occurs in the form of rapidly repeated contractions, as in "chattering teeth." This is rare apart from general conditions, though cases are on record, usually in women late in life, in whom this isolated clonic spasm of the muscles of the jaw has been found. In another form of clonic spasm sometimes seen in chorea there are forcible single contractions. Gowers mentions an instance of its occurrence as an isolated affection.

(*c*) Gustatory.—Complete or partial loss of the sense of taste over the anterior two-thirds of the tongue has been supposed by some to follow paralysis of the fifth nerve. There are two views concerning the course of the fibres that carry gustatory impulse from this part of the tongue. According to some they take a devious path, passing with the chorda tympani to the geniculate ganglion, thence by the great superficial petrosal nerve to Meckel's ganglion, and this they leave to reach the maxillary nerve, which they follow through the trigeminal nerve to the brain. A study of clinical cases of disease of the fifth nerve has led to this view. It seems more probable, however, from the fact that a large number of the trigeminal neurectomies are not followed by loss of taste, that the fibres pass to the brain directly from the geniculate ganglion by the nervus intermedius of Wrisberg. Possibly there may be more than one course for these fibres.

The *diagnosis* of disease of the trifacial nerve is rarely difficult. It must be remembered that the preliminary pain and hyperæsthesia are sometimes mistaken for ordinary neuralgia. The loss of sensation and the palsy of the muscles of mastication are readily determined.

Treatment.—When the pain is severe morphia may be required and local applications are useful. If there is a suspicion of syphilis, appropriate treatment should be given. Faradization is sometimes beneficial.

FACIAL NERVE

Paralysis (*Bell's Palsy*).—ETIOLOGY.—The facial or seventh may be paralyzed by (*a*) lesions of the cortex—supranuclear palsy; (*b*) lesions of the nucleus itself; or (*c*) involvement of the nerve trunk in its tortuous course within the pons and through the wall of the skull.

(*a*) *Supranuclear paralysis,* due to lesion of the cortex or of the facial fibres in the corona radiata or internal capsule, is, as a rule, associated with hemiplegia. It may be caused by tumors, abscess, chronic inflammation, or softening in the cortex or in the region of the internal capsule. It is distinguished from the peripheral form by well marked characters—the persistence of the normal electrical excitability of both nerves and muscles and the frequent absence of involvement of the upper branches of the nerve, so that the orbicularis palpebrarum, frontalis, and corrugator muscles are spared. In rare instances these muscles are paralyzed. In this form the voluntary movements are more impaired than the emotional. Isolated paralysis—monoplegia facialis—due to involvement of the cortex or of the fibres in their path to the nucleus, is uncommon. In the great majority of cases supranuclear facial paralysis is part of a hemiplegia. Paralysis is on the same side as that of the arm and leg because the facial muscles bear precisely the same relation to the cortex as the spinal muscles. The nuclei of origin on either side of the middle line in the medulla are united by decussating fibres with the cortical centre on the opposite side (see Fig. 18). A few fibres reach the nucleus from the cerebral cortex of the same side, and this uncrossed path may innervate the upper facial muscles.

(*b*) The *nuclear paralysis* caused by lesions of the nerve centres in the medulla is not common alone; but is seen occasionally in tumors, chronic softening, and haemorrhage. It may be involved in anterior polio-myelitis. In diphtheria this centre may also be attacked. The symptoms are practically similar to those of an affection of the nerve fibre itself—infranuclear paralysis.

(*c*) *Involvement of the Nerve Trunk.*—Paralysis may result from:

(1) Involvement of the nerve as it passes through the pons—that is, between its nucleus in the floor of the fourth ventricle and the point of emergence in the postero-lateral aspect of the pons. The specially interesting feature in connection with involvement of this part is the production of what is called alternating or *crossed paralysis,* the face being involved on the same side as the lesion, and the arm and leg on the opposite side, since the motor path is involved above the point of decussation in the medulla (Fig. 18). This occurs only when the lesion is in the lower section of the pons. A lesion in the upper half of the pons involves the fibres not of the outgoing nerve on the same side, but of the fibres from the hemispheres before they have crossed to the nucleus of the opposite side. In this case there would of course be, as in hemiplegia, paralysis of the face and limbs on the side opposite to the lesion. The palsy, too, would resemble the cerebral form, involving only the lower fibres of the facial nerve.

(2) The nerve may be involved at its point of emergence by tumors, particularly by the cerebello-pontine growths, by gummata, meningitis, or occasionally it may be injured in fracture of the base.

(3) In passing through the Fallopian canal the nerve may be involved in disease of the ear, particularly by caries of the bone in otitis media. This is a common cause in children. I have seen two instances follow otitis in puerperal fever.

(4) As the nerve emerges from the styloid foramen it is exposed to injuries and blows which not infrequently cause paralysis. The fibres may be cut in the removal of tumors in this region, or the paralysis may be caused by pressure of the forceps in an instrumental delivery.

(5) Exposure to cold is the most common cause of facial paralysis (Bell's palsy), inducing a neuritis of the nerve within the Fallopian canal. Reik believes that in most of these cases there is an acute otitis media from which the nerve is involved.

(6) Syphilis is not an infrequent cause, and the paralysis may appear early with the secondary symptoms.

(7) It may occur in association with herpes.

Facial diplegia is a rare condition occasionally found in affections at the base of the brain, lesions in the pons, simultaneous involvement of the nerves in ear-disease, and in diphtheritic paralysis. Disease of the nuclei or symmetrical involvement of the cortex might also produce it. It may occur as a congenital affection. H. M. Thomas has described two cases in one family.

Symptoms.—In the peripheral facial paralysis all the branches of the nerve are involved. The face on the affected side is immobile and can neither be moved at will nor participate in any emotional movements. The skin is smooth and the wrinkles are effaced, a point particularly noticeable on the forehead of elderly persons. The eye can not be closed, the lower lid droops, and the eye waters. On the affected side the angle of the mouth is lowered, and in drinking the lips are not kept in close apposition to the glass, so that the liquid is apt to run out. In smiling or laughing the contrast is most striking, as the affected side does not move, which gives a curious unequal appearance to the two sides of the face. The eye can not be closed nor can the forehead be wrinkled. In long standing cases, when the reaction of degeneration is present, if the patient tries to close the eyes while looking fixedly at an object the lids on the sound side close firmly, but on the paralyzed side there is only a slight inhibitory droop of the upper lid, and the eye is turned upward and outward by the inferior oblique. On asking the patient to show his upper teeth, the angle of the mouth is not raised. In all these movements the face is drawn to the sound side by the action of the muscles. Speaking may be slightly interfered with, owing to the imperfection in the formation of the labial sounds. Whistling can not be performed. In chewing the food, owing to the paralysis of the buccinator, particles collect on the affected side. The paralysis of the nasal muscles is seen on asking the patient to sniff. Owing to the fact that the lips are drawn to the sound side, the tongue, when protruded, looks as if it were pushed to the paralyzed side; but on taking its position from the incisor teeth, it will be found to be in the middle line. The reflex movements are lost in this peripheral form. It is usually stated that the palate is partially paralyzed on the same side and that the

uvula deviates. Both Gowers and Hughlings Jackson deny the existence of this involvement in the great majority of cases, and Horsley and Beevor have shown that these parts are innervated by the accessory nerve to the vagus.

The *sensory functions* of the facial nerve, to which much attention has been paid ~~of late~~ by Cushing, Mills and others, are ministered to by the geniculate ganglion, the intermediary nerve of Wrisberg, and the chorda tympani, which last has chiefly gustatory functions. It seems likely that deep sensibility with sense of pressure, position and passive movement runs in a separate afferent system in the motor nerve of the face. Cutaneous sensibility, both epicritic, by which we localize light touch, and protopathic, by which we recognize degrees of heat and cold, is not ministered to by the facial nerve proper. There are observations that would indicate, however, that the anterior part of the tongue and possibly a little strip of the skin of the auricle have a vestigial supply from this nerve.

When the nerve is involved within the canal between the genu and the origin of the chorda tympani, the sense of taste is lost in the anterior part of the tongue on the affected side. When the nerve is damaged outside the skull the sense of taste is unaffected. Hearing is often impaired in facial paralysis, most commonly by preceding ear disease. The paralysis of the stapedius muscle may lead to increased sensitiveness to musical notes. Herpes is sometimes associated with facial paralysis. Pain is not common, but there may be neuralgia about the ear. The face on the affected side may be swollen.

The *electrical reactions,* which are those of a peripheral palsy, have considerable importance from a prognostic standpoint. Erb's rules are as follows: If there is no change, either faradic or galvanic, the prognosis is good and recovery takes place in from fourteen to twenty days. If the faradic and galvanic excitability of the nerve is only lessened and that of the muscle increased to the galvanic current and the contraction formula altered (the contraction sluggish AC<KC), the outlook is relatively good and recovery will probably take place in from four to six weeks; occasionally in from eight to ten. When the reaction of degeneration is present—that is, if the faradic and galvanic excitability of the nerves and the faradic excitability of the muscles are lost and the galvanic excitability of the muscle is quantitatively increased and qualitatively changed, and if the mechanical excitability is altered—the prognosis is relatively unfavorable and the recovery may not occur for two, six, eight, or even fifteen months.

Course.—The course of facial paralysis is usually favorable. The onset in the form following cold is very rapid, developing perhaps within twenty-four hours, but rarely is the paralysis permanent. Now and again the paralysis never disappears; ~~after four years~~ I have seen only slight recovery. Recurring attacks have been described; Sinkler mentions five. ~~On the other~~ hand, in the paralysis from injury, as by a blow on the mastoid process, the condition may remain. When permanent, the muscles are entirely toneless. In some instances contracture develops as the voluntary power returns, and the natural folds and the wrinkles on the affected side may be deepened, so that on looking at the face one at first may have the impression that the affected side is the sound one. This is corrected at once on asking the patient to smile, when it is seen which side of the face has the most active movement.

Aretæus noted the difficulty sometimes experienced in determining which side was affected until the patient spoke or laughed.

Diagnosis.—The diagnosis of facial paralysis is usually easy. The distinction between the peripheral and central form is based on facts already mentioned.

Treatment.—In the cases which result from cold and are probably due to neuritis within the bony canal, hot applications first should be made; subsequently the thermo-cautery may be used lightly at intervals of a day or two over the mastoid process, or small blisters applied. If the ear is diseased, free discharge for the secretion should be obtained. The galvanic current may be employed to keep up the nutrition of the muscles. The positive pole should be placed behind the ear, the negative one along the zygomatic and other muscles. The application can be made daily for a quarter of an hour and the patient can readily be taught to make it himself before a looking glass. Massage in the course of the nerve and of the muscles of the face is also useful. A course of iodide of potassium may be given even when there is no indication of syphilis.

In those cases in which the nerve has been destroyed by an injury, during an operation or from disease, and when there has been no evidence of returning function after keeping up the electric treatment for a few months, a nerve anastomosis should be performed. For this purpose either the spinal accessory or the hypoglossal nerve may be used. Though the normal conditions may never be completely regained after such an operation, the motor power will be largely restored to the paralyzed muscles and the obtrusive deformity greatly lessened. This procedure, based on the results of physiological experimentation, makes one of the most striking of modern operations.

Spasm.—The spasm may be limited to a few or involve all the muscles innervated by the facial nerve, and may be unilateral or bilateral.

It is known also by the name of mimic spasm or of convulsive tic. Several different affections are usually considered under the name of facial or mimic spasm, but we shall here speak only of the simple spasm of the facial muscles, either primary or following paralysis, and shall not include the cases of habit spasm in children, or the *tic convulsif* of the French.

Gowers recognizes two classes—one in which there is an organic lesion, and an idiopathic form. It is thought to be due also to reflex causes, such as the irritation from carious teeth or the presence of intestinal worms. The disease usually occurs in adults, whereas the habit spasm and the *tic convulsif* of the French, often confounded with it, are most common in children. True mimic spasm occasionally comes on in childhood and persists. In the case of a school-mate the affection was marked as early as the eleventh or twelfth year and still continues. When the result of organic disease, there has usually been a lesion of the centre in the cortex, as in the case reported by Berkley, or pressure on the nerve at the base of the brain by aneurism or tumor.

Symptoms.—The spasm may involve only the muscles around the eye—blepharospasm—in which case there is constant, rapid, quick action of the orbicularis palpebrarum, which, in association with photophobia, may be tonic in character. More commonly the spasm affects the lateral facial muscles with those of the eye, and there is constant twitching of the side of the face with partial closure of the eye. The frontalis is rarely involved. In aggravated

cases the depressors of the angle of the mouth, the levator menti, and the platysma myoides are affected. This spasm is confined to one side of the face in a majority of cases, though it may extend and become bilateral. It is increased by emotional causes and by voluntary movements of the face. As a rule, it is painless, but there may be tender points over the course of the fifth nerve, particularly the supraorbital branch. Tonic spasm of the facial muscle may follow paralysis, and is said to result occasionally from cold.

The outlook in facial spasm is always dubious. A majority of the cases persist for years and are incurable.

TREATMENT.—Sources of irritation should be looked for and removed. When a painful spot is present over the fifth nerve, blistering or the application of the thermo-cautery may relieve it. Hypodermic injections of strychnia may be tried, but are of doubtful benefit. Weir Mitchell recommends the freezing of the cheek for a few minutes daily or every second day with the spray, and this, in some instances, is beneficial. Often the relief is transient: the cases return, and at every clinic may be seen half a dozen or more of such patients who have run the gamut of all measures without material improvement. Severe cases may require surgical interference. The nerve may be divided near the stylomastoid foramen and an anastomosis made between it and the spinal accessory.

AUDITORY NERVE

The eighth, known also as *portio mollis* of the seventh pair, passes from the ear through the internal auditory meatus, and in reality consists of two separate nerves—the cochlear and vestibular roots. These two roots have entirely different functions, and may therefore be best considered separately. The cochlear nerve is the one connected with the organ of Corti, and is concerned in hearing. The vestibular nerve is connected with the vestibule and semicircular canals, and has to do with the maintenance of equilibrium.

The Cochlear Nerve

The cortical centre for hearing is in the temporo-sphenoidal lobe. Primary disease of the auditory nerve in its centre or intracranial course is uncommon. More frequently the terminal branches are affected within the labyrinth.

Affection of the Cortical Centre.—The superior temporal gyrus represents the centre for hearing. In man destruction of this gyrus on the left side results in word-deafness, which may be defined as an inability to understand the meaning of words, though they may still be heard as sounds. The central auditory path extending to the cortical centre from the terminal nuclei of the cochlear nerve may be involved and produce deafness. This may result from involvement of the lateral lemniscus, from the presence of a tumor in the corpora quadrigemina, especially if it involve the posterior quadrigeminal bodies, from a lesion of the internal geniculate body, or it may be associated with a lesion of the internal capsule.

Lesions of the nerve at the base of the brain may result from the pressure of tumors, meningitis (particularly the cerebro-spinal form), hæmorrhage, or traumatism. A primary degeneration of the nerve may occur in locomotor ataxia. Primary disease of the terminal nuclei of the cochlear nerve (nucleus

nervi cochlearis dorsalis and nucleus nervi cochlearis ventralis) is rare. By far the most interesting form results from epidemic cerebro-spinal meningitis, in which the nerve is frequently involved, causing permanent deafness. In young children the condition results in deaf-mutism.

Internal Ear.—In a majority of the cases associated with auditory nerve symptoms the lesion is in the internal ear, either primary or the result of extension of disease of the middle ear. Two groups of symptoms may be produced—hyperæsthesia and irritation, and diminished function or nervous deafness.

(*a*) Hyperæsthesia and Irritation.—This may be due to altered function of the centre as well as of the nerve ending. True hyperæsthesia—hyperacusis—is a condition in which sounds, sometimes even those inaudible to other persons, are heard with great intensity. It occurs in hysteria and occasionally in cerebral disease. As already mentioned, in paralysis of the stapedius low notes may be heard with intensity. In dysæsthesia, or dysacusis, ordinary sounds cause an unpleasant sensation, as commonly happens in connection with headache, when ordinary noises are badly borne.

Tinnitus aurium is a term employed to designate certain subjective sensations of ringing, roaring, tickling, and whirring noises in the ear. It is a very common and often a distressing symptom. It is associated with many forms of ear disease and may result from pressure of wax on the drum. It is rare in organic disease of the central connections of the nerve. Sudden intense stimulation of the nerve may cause it. A form not uncommonly met with in medical practice is that in which the patient hears a continual *bruit* in the ear, and the noise has a systolic intensification, usually on one side. I have twice been consulted by physicians for this condition under the belief that they had an internal aneurism. A systolic murmur may be heard occasionally on auscultation. It occurs in conditions of anæmia and neurasthenia. Subjective noises in the ear may precede an epileptic seizure and are sometimes present in migraine. In whatever form tinnitus exists, though slight and often regarded as trivial, it occasions great annoyance and often mental distress, and has even driven patients to suicide.

The *diagnosis* is readily made; but it is often extremely difficult to determine upon what condition the tinnitus depends. The relief of constitutional states, such as anæmia, neurasthenia, or gout, may result in cure. A careful local examination of the ear should always be made. One of the most worrying forms is the constant clicking, sometimes audible many feet away from the patient, and due probably to clonic spasm of the muscles connected with the Eustachian tube or of the levator palati. The condition may persist for years unchanged, and then disappear suddenly. The pulsating forms of tinnitus, in which the sound is like that of a systolic *bruit*, are almost invariably subjective, and it is very rare to hear anything with the stethoscope. It is to be remembered that in children there is a systolic brain murmur, best heard over the ear, and in some instances appreciable in the adult.

(*b*) Diminished Function or Nervous Deafness.—In testing for nervous deafness, if the tuning fork can not be heard when placed near the meatus, but the vibrations are audible by placing the foot of the tuning fork against the temporal bone, the conclusion may be drawn that the deafness is not due to involvement of the nerve. The vibrations are conveyed through the tem-

poral bone to the cochlea and vestibule. The watch may be used for the same purpose, and if the meatus is closed and the watch is heard better in contact with the mastoid process than when opposite the open meatus, the deafness is probably not nervous. Disturbance of the function of the auditory nerve is not a very frequent symptom in brain disease, but in all cases the function of the nerve should be carefully tested.

The Vestibular Nerve

The most frequent symptoms met with in association with disease of the vestibular nerve and its central connections are vertigo, nystagmus, and loss of coördination of the muscles of the head, neck, and eyes.

Auditory Vertigo—Ménière's Disease.—In 1861 Ménière, a French physician, described an affection characterized by noises in the ear, vertigo (which might be associated with loss of consciousness), vomiting, and, in many cases, progressive loss of hearing. Bárány, of Vienna, has thrown much light on this subject. He groups the conditions in which the labyrinth may be affected under the following heads: (*a*) Acute infectious diseases, influenza, cerebro-spinal meningitis, mumps, etc. (*b*) Chronic infectious diseases, syphilis particularly. (*c*) Constitutional conditions and auto-intoxications. Hæmorrhage into the labyrinth (in leukæmia, purpura hæmorrhagica, pernicious anæmia); chlorosis, thyroid intoxications, arterio-sclerosis, etc. (*d*) Tumors and diseases of the central nervous system; tumors of the acoustic nerve, cerebellum, pons, and fourth ventricle, meningitis, cerebellar abscess, multiple sclerosis, tabes, etc. (*e*) Traumatic injuries, fracture of the base, etc. (*f*) Hereditary degenerative diseases and malformations of the internal ear. (*g*) Intoxications, alcohol, nicotine, quinine, salicylic acid group, arsenic (salvarsan?).

Symptoms.—The attack usually sets in suddenly with a buzzing noise in the ears and the patient feels as if he was reeling or staggering. He may feel himself to be reeling, or the objects about him may seem to be turning, or the phenomena may be combined. The attack is often so abrupt that the patient falls, though, as a rule, he has time to steady himself by grasping some neighboring object. Consciousness is generally maintained, but may be momentarily lost. Ocular symptoms are usually present. Jerking of the eyeballs, or nystagmus, occurs. The patient becomes pale and nauseated, a clammy sweat breaks out on the face, and vomiting may follow. The duration of the attack varies greatly. At times it may be very short, but it usually causes the patient to lie quietly for some time, as any movement of the head brings on another attack.

Labyrinthine vertigo is usually paroxysmal, coming on at irregular intervals, sometimes of weeks or months; or several attacks may occur in a day.

The disturbances of equilibrium, including the vertigo, are dependent upon a disturbance of the functions of the vestibular nerve or of the organs with which this nerve is connected, either in its peripheral distribution or by means of its central connection. The auditory symptoms often accompanying it are doubtless always due to involvement of the cochlear nerve or its peripheral or central connections.

Diagnosis.—The combination of tinnitus with giddiness, with or without

gastric disturbance, is sufficient to establish a diagnosis. There are other forms of vertigo from which it must be distinguished. The form known as gastric vertigo, which is associated with dyspepsia and occurs most commonly in persons of middle age, is, as a rule, readily distinguished by the absence of tinnitus or evidences of disturbance in the function of the auditory nerve. This variety of vertigo is much less common than Trousseau's description would lead us to believe. It is important to note the close connection of vertigo with ocular defects.

The cardio-vascular vertigo, one of the most common forms, occurs in cases of valvular disease, particularly aortic insufficiency, and as frequently in arterio-sclerosis.

Aural vertigo must be carefully distinguished from attacks of *petit mal*, or, indeed, of definite epilepsy. It is rare in *petit mal* to have noises in the ear or actual giddiness, but in the aura preceding an epileptic attack the patient may feel giddy. Giddiness and transient loss of consciousness may be associated with organic disease of the brain, more particularly with tumor. Vomiting also may be present. A careful investigation will usually lead to a correct diagnosis. Bárány's special tests for the functional activity of the vestibular nerve are of great use in the hands of a skilled observer, particularly his caloric test (irrigation of the external meatus with cold or warm water, and observing its effect on the production of nystagmus).

Prognosis.—The outlook in Ménière's disease is uncertain. While many cases recover completely, in others deafness results and the attacks recur at shorter intervals. In aggravated cases the patient constantly suffers from vertigo, and may even be confined to his bed.

Treatment.—Bromide of potassium, in 20 grain (1.3 gm.) doses three times a day, is sometimes beneficial. If there is a history of syphilis the iodides should be administered. The salicylates are recommended, and Charcot advises quinine to cinchonism. In cases in which there is increase in the arterial tension nitroglycerin may be given, at first in very small doses, but increasing gradually. It is not specially valuable in Ménière's disease, but in the cases of giddiness in middle aged men and women associated with arterio-sclerosis it sometimes acts very satisfactorily. Correction of errors of refraction is sometimes followed by prompt relief of the vertigo.

Endemic Paralytic Vertigo.—In parts of Switzerland and France there is a remarkable form of vertigo described by Gerlier, which is characterized by attacks of paretic weakness of the extremities, falling of the eyelids, remarkable depression, but with retention of consciousness. It occurs also in northern Japan, where Miura says it develops paroxysmally among the farm laborers of both sexes and all ages. It is known there as *kubisagari*.

GLOSSO-PHARYNGEAL NERVE

(*Nervus glossopharyngeus*)

The ninth nerve contains both motor and sensory fibres and is also a nerve of the special sense of taste to the tongue. It supplies, by its motor branches, the stylo-pharyngeus and the middle constrictor of the pharynx. The sensory fibres are distributed to the upper part of the pharynx.

Symptoms.—Of nuclear disturbance we know very little. The pharyngeal symptoms of bulbar paralysis are probably associated with involvement of the nuclei of this nerve. Lesion of the nerve trunk itself is rare, but it may be compressed by tumors or involved in meningitis. Disturbance of the sense of taste may result from loss of function of this nerve, in which case it is chiefly in the posterior part of the tongue and soft palate.

The general disturbances of the sense of taste may here be briefly referred to. Loss of the sense of taste—*ageusia*—may be caused by disturbance of the peripheral end organs, as in affections of the mucosa of the tongue. This is very common in the dry tongue of fever or the furred tongue of dyspepsia, under which circumstances, as the saying is, everything tastes alike. Strong irritants, too, such as pepper, tobacco, or vinegar, may dull or diminish the sense of taste. Complete loss may be due to involvement of the nerves either in their course or in the centres. Perversion of the sense of taste—*parageusis*—is rarely found, except as an hysterical manifestation and in the insane. Increased sensitiveness is still more rare. There are occasional subjective sensations of taste, occurring as an aura in epilepsy or as part of the hallucinations in the insane.

To test the sense of taste the patient's eyes should be closed and small quantities of various substances applied to the protruded tongue. The sensation should be perceived before the tongue is withdrawn. The following are the most suitable tests: For bitterness, quinine; for sweetness, a strong solution of sugar or saccharin; for acidity, vinegar; and for the saline test, common salt. One of the most important tests is the feeble galvanic current, which gives the well-known metallic taste.

PNEUMOGASTRIC NERVE

(*Nervus vagus*)

The tenth nerve has an important and extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach. The nerve may be involved at its nucleus along with the spinal accessory and the hypoglossal, forming what is known as bulbar paralysis. It may be compressed by tumors or aneurism, or in the exudation of meningitis, simple or syphilitic. In its course in the neck the trunk may be involved by tumors or in wounds. It has been tied in ligature of the carotid, and has been cut in the removal of deep-seated tumors. The trunk may be attacked by neuritis.

The affections of the vagus are best considered in connection with the distribution of the separate nerves.

Pharyngeal Branches.—In combination with the glosso-pharyngeal the branches from the vagus form the pharyngeal plexus, from which the muscles and mucosa of the pharynx are supplied. In *paralysis* due to involvement of this either in the nuclei, as in bulbar paralysis, or in the course of the nerve, as in diphtheritic neuritis, there is difficulty in swallowing and the food is not passed on into the œsophagus. If the nerve on one side only is involved the deglutition is not much impaired. In these cases the particles of food frequently pass into the larynx, and, when the soft palate is involved, into the posterior nares.

Spasm of the pharynx is always a functional disorder, usually occurring in hysterical and nervous people. Gowers mentions a case of a gentleman who could not eat unless alone, on account of the inability to swallow in the presence of others from spasm of the pharynx. This spasm is a well marked feature in hydrophobia, and it occurs also in pseudo-hydrophobia.

Laryngeal Branches.—The superior laryngeal nerve supplies the mucous membrane of the larynx above the cords and the crico-thyroid muscle. The inferior or recurrent laryngeal curves around the arch of the aorta on the left side and the subclavian artery on the right passes along the trachea and supplies the mucosa below the cords and all the muscles of the larynx except the crico-thyroid and the epiglottidean. Experiments have shown that these motor nerves of the pneumogastric are all derived from the spinal accessory. The remarkable course of the recurrent laryngeal nerves renders them liable to pressure by tumors within the thorax, particularly by aneurism. The following are the most important forms of paralysis:

(*a*) Bilateral Paralysis of the Abductors.—In this condition the posterior crico-arytenoids are involved and the glottis is not opened during inspiration. The cords may be close together in the position of phonation, and during inspiration may be brought even nearer together by the pressure of air, so that there is only a narrow chink through which the air whistles with a noisy stridor. This dangerous form of laryngeal paralysis occurs occasionally as a result of cold, or may follow a laryngeal catarrh. The posterior muscles have been found degenerated when the others were healthy. The condition may be produced by pressure upon both vagi, or upon both recurrent nerves. As a central affection it occurs in tabes and bulbar paralysis, but may be seen also in hysteria. The characteristic symptoms are inspiratory stridor with unimpaired phonation. Possibly, as Gowers suggests, many cases of so-called hysterical spasm of the glottis are in reality abductor paralysis.

(*b*) Unilateral Abductor Paralysis.—This frequently results from the pressure of tumors or involvement of one recurrent nerve. Aneurism is by far the most common cause, though on the right side the nerve may be involved in thickening of the pleura. The symptoms are hoarseness or roughness of the voice, such as is so common in aneurism. Dyspnœa is not often present. The cord on the affected side does not move in inspiration. Subsequently the adductors may also become involved, in which case the phonation is still more impaired.

(*c*) Adductor Paralysis.—This results from involvement of the lateral crico-arytenoid and the arytenoid muscle itself. It is common in hysteria, particularly of women, and causes the hysterical aphonia, which may come on suddenly. It may result from catarrh of the larynx or from overuse of the voice. In laryngoscopic examination it is seen, on attempting phonation, that there is no power to bring the cords together.

(*d*) Spasm of the Muscles of the Larynx.—In this the adductor muscles are involved. It is not an uncommon affection in children, and has already been referred to as laryngismus stridulus. Paroxysmal attacks of laryngeal spasm are rare in the adult, but cases are described in which the patient, usually a young girl, wakes at night in an attack of intense dyspnœa, which may persist long enough to produce cyanosis. Liveing states that they may replace attacks of migraine. They occur in a characteristic form in loco-

motor ataxia, forming the so-called laryngeal crises. There is a condition known as spastic aphonia, in which, when the patient attempts to speak, phonation is completely prevented by a spasm.

Disturbance of the sensory nerves of the larynx is rare.

(*e*) Anæsthesia may occur in bulbar paralysis and in diphtheritic neuritis —a serious condition, as portions of food may enter the windpipe. It is usually associated with dysphagia and is sometimes present in hysteria. Hyperæsthesia of the larynx is rare.

Cardiac Branches.—The cardiac plexus is formed by the union of branches of the vagi and of the sympathetic nerves. The vagus fibres subserve motor, sensory, and probably trophic functions.

Motor.—The fibres which inhibit, control, and regulate the cardiac action pass in the vagi. Irritation may produce slowing of the action. Czermak could slow or even arrest the heart's action for a few beats by pressing a small tumor in his neck against one pneumogastric nerve, and it is said that the same can be produced by forcible bilateral pressure on the carotid canal. There are instances in which persons appear to have had voluntary control over the action of the heart. Cheyne mentions the case of Colonel Townshend, "who could die or expire when he pleased, and yet by an effort or somehow come to life again, which it seems he had sometimes tried before he had sent for us." Retardation of the heart's action has also followed accidental ligature of one vagus. Irritation of the nuclei may also be accompanied with a neurosis of this nerve. On the other hand, when there is complete paralysis of the vagi, the inhibitory action may be abolished and the acceleratory influences have full sway. The heart's action is then greatly increased. This is seen in some instances of diphtheritic neuritis and in involvement of the nerve by tumors, or its accidental removal or ligature. Complete loss of function of one vagus, however, may not be followed by any symptoms.

Sensory symptoms on the part of the cardiac branches are very varied. Normally, the heart's action proceeds regularly without the participation of consciousness, but the unpleasant feelings and sensations of palpitation and pain are conveyed to the brain through this nerve. How far the fibres of the pneumogastric are involved in angina it is impossible to say. The various disturbances of sensation are described under the cardiac neuroses.

Pulmonary Branches.—We know very little of the pulmonary branches of the vagi. The motor fibres are stated to control the action of the bronchial muscles, and it has long been held that asthma may be a neurosis of these fibres. The various alterations in the respiratory rhythm are probably due more to changes in the centre than in the nerves themselves.

Gastric and Œsophageal Branches.—The muscular movements of these parts are presided over by the vagi and vomiting is induced through them, usually reflexly, but also by direct irritation, as in meningitis. Spasm of the œsophagus generally occurs with other nervous phenomena. Gastralgia may sometimes be due to cramp of the stomach, but is more commonly a sensory disturbance of this nerve, due to direct irritation of the peripheral ends, or is a neuralgia of the terminal fibres. Hunger is said to be a sensation aroused by the pneumogastric, and some forms of nervous dyspepsia probably depend upon disturbed function of this nerve. The severe gastric crises which occur in locomotor ataxia are due to central irritation of the nuclei.

SPINAL ACCESSORY NERVE

(*Nervus accessorius*)

Paralysis.—The smaller or internal part of this nerve joins the vagus and is distributed through it to the laryngeal muscles. The larger external part is distributed to the sterno-mastoid and trapezius muscles.

The nuclei of the nerve, particularly of the accessory part, may be involved in bulbar paralysis. The nuclei of the external portion, situated as they are in the cervical cord, may be attacked in progressive degeneration of the motor nuclei of the cord. The nerve may be involved in the exudation of meningitis, or be compressed by tumors, or in caries. The *symptoms* of paralysis of the accessory portion which joins the vagus have already been given in the account of the palsy of the laryngeal branches of the pneumogastric. Disease or compression of the external portion is followed by paralysis of the sterno-mastoid and of the trapezius on the same side. In paralysis of one sterno-mastoid the patient rotates the head with difficulty to the opposite side, but there is no torticollis, though in some cases the head is held obliquely. As the trapezius is supplied in part from the cervical nerves, it is not completely paralyzed, but the portion which passes from the occipital bone to the acromion is functionless. The paralysis of the muscle is well seen when the patient draws a deep breath or shrugs the shoulders. The middle portion of the trapezius is also weakened, the shoulder droops a little, and the angle of the scapula is rotated inward by the action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is impaired, for the trapezius does not fix the scapula as a point from which the deltoid can work.

In progressive muscular atrophy we sometimes see bilateral paralysis of these muscles. Thus, if the sterno-mastoids are affected, the head tends to fall back; when the trapezii are involved, it falls forward, a characteristic attitude of the head in many cases of progressive muscular atrophy. Gowers suggests that lesions of the accessory in difficult labor may account for those cases in which during the first year of life the child has great difficulty in holding up the head. In children this drooping of the head is an important symptom in cervical meningitis, the result of caries.

The TREATMENT of the condition depends much upon the cause. In the central nuclear atrophy but little can be done. In paralysis from pressure the symptoms may gradually be relieved. The paralyzed muscles should be stimulated by electricity and massage.

Accessory Spasm (*Torticollis; Wryneck*).—The forms of spasm affecting the cervical muscles are best considered here, as the muscles supplied by the accessory are chiefly, though not solely, responsible for the condition. The following forms may be described in this section:

(*a*) CONGENITAL TORTICOLLIS.—This condition, also known as fixed torticollis, depends upon the shortening and atrophy of the sterno-mastoid on one side. It occurs in children and may not be noticed for several years on account of the shortness of the neck, the parents often alleging that it has only recently come on. It affects the right side almost exclusively. A remarkable circumstance in connection with it is the existence of facial asymmetry noted by Wilks, which appears to be an essential part of this congenital

form. In congenital wryneck the sterno-mastoid is shortened, hard and firm, and in a condition of more or less advanced atrophy. This must be distinguished from the local thickening in the sterno-mastoid due to rupture, which may occur at the time of birth and produce an induration or muscle callus. Although the sterno-mastoid is almost always affected, there are rare cases in which the fibrous atrophy affects the trapezius. This form of wryneck in itself is unimportant, since it is readily relieved by tenotomy, but Golding-Bird states that the facial asymmetry persists, or, indeed, may, as shown by photographs ~~in my case~~, become more evident. With reference to the pathology, ~~of the affection~~, Golding-Bird concludes that the facial asymmetry and the torticollis are integral parts of one affection which has a central origin, and is the counterpart in the head and neck of infantile paralysis with talipes in the foot.

(*b*) Spasmodic Wryneck.—Two varieties of this spasm occur, the tonic and the clonic, which may alternate in the same case; or, as is most common, they are separate and remain so from the outset. The disease is most frequent in adults and, according to Gowers, more common in females. In America it is certainly more frequent in males. In females it may be an hysterical manifestation. There may be a marked neurotic family history, but it is usually impossible to fix upon any definite etiological factor. Some cases have followed cold; others a blow. Brissaud has described what he calls mental torticollis. It is usually met with in neurasthenic patients and in elderly persons, and consists of a clonic spasm of the rotators of the head.

The *symptoms* are well defined. In the tonic form the contracted sterno-mastoid draws the occiput toward the shoulder of the affected side; the chin is raised, and the face rotated to the other shoulder. The sterno-mastoid may be affected alone or in association with the trapezius. When the latter is implicated the head is depressed still more toward the same side. In long-standing cases these muscles are prominent and very rigid. There may be some curvature of the spine, the convexity of which is toward the sound side. The cases in which the spasm is clonic are much more distressing and serious. The spasm is rarely limited to a single muscle. The sterno-mastoid is almost always involved and rotates the head so as to approximate the mastoid process to the inner end of the clavicle, turning the face to the opposite side and raising the chin. When with this the trapezius is affected, the depression of the head toward the same side is more marked. The head is drawn somewhat backward; the shoulder, too, is raised by its action. According to Gowers, the splenius is associated with the sterno-mastoid about half as frequently as the trapezius. Its action is to incline the head and rotate it slightly toward the same side. Other muscles may be involved, such as the scalenus and platysma myoides; and in rare cases the head may be rotated by the deep cervical muscles, the rectus and obliquus. There are cases in which the spasm is bilateral, causing a backward movement—the retro-collic spasm. This may be either tonic or clonic, and in extreme cases the face is horizontal and looks upward.

These clonic contractions may come on without warning, or be preceded for a time by irregular pains or stiffness of the neck. The jerking movements recur every few moments, and it is impossible to keep the head still for more than a minute or two. In time the muscles undergo hypertrophy and may be

distinctly larger on one side than the other. In some cases the pain is considerable; in others there is simply a feeling of fatigue. The spasms cease during sleep. Emotion, excitement, and fatigue increase them. The spasm may extend from the muscles of the neck and involve those of the face or the arms.

The disease varies much in its course. Cases occasionally get well, but the great majority of them persist, and, even if temporarily relieved, the disease frequently recurs. The affection is usually regarded as a functional neurosis, but it is possibly due to disturbance of the cortical centres presiding over the muscles.

Treatment.—Temporary relief is sometimes obtained; a permanent cure is exceptional. Various drugs have been used, but rarely with benefit. Occasionally, large doses of bromide will lessen the intensity of the spasm. Morphia, subcutaneously, has been successful in some reported cases, but there is the great danger of establishing the morphia habit. Galvanism may be tried. Counter-irritation is probably useless. Fixation of the head mechanically can rarely be borne by the patient. These obstinate cases fall ultimately into the hands of the surgeon, and the operations of stretching, division, and excision of the accessory nerve and division of the muscles have been tried. Temporary relief may follow, but, as a rule, the condition returns. Risien Russell thinks that resection of the posterior branches of the upper cervical nerves is most likely to give relief, and this has been done by Keen and others.

(*c*) The NODDING SPASM of children may here be mentioned as involving chiefly the muscles innervated by the accessory nerve. It may be a simple trick, a form of habit spasm, or a phenomenon of epilepsy (E. nutans), in which case it is associated with transient loss of consciousness. A similar nodding spasm may occur in older children. In women it sometimes occurs as an hysterical manifestation, commonly as part of the so-called salaam convulsion.

HYPOGLOSSAL NERVE

This is the motor nerve of the tongue and for most of the muscles attached to the hyoid bone. Its cortical centre is probably the lower part of the anterior central gyrus.

Paralysis.—(*a*) CORTICAL LESION.—The tongue is often involved in hemiplegia, and the paralysis may result from a lesion of the cortex itself, or of the fibres as they pass to the medulla. It does not occur alone and is considered with hemiplegia. There is this difference, however, between the cortical and other forms, that the muscles on both sides of the tongue may be more or less affected but do not waste, nor are their electrical reactions disturbed.

(*b*) NUCLEAR and INFRA-NUCLEAR lesions of the hypoglossal result from slow progressive degeneration, as in bulbar paralysis or in locomotor ataxia; occasionally there is acute softening from obstruction of the vessels. The nuclei of both nerves are usually affected together, but may be attacked separately. Trauma and lead poisoning have also been assigned as causes. The fibres may be damaged by a tumor, and at the base by meningitis; or the nerve is sometimes involved in the condylar foramen by disease of the skull. It may be involved in its course in a scar, as in Birkett's case, or compressed by a

tumor in the parotid region. As a result, there is loss of function in the nerve fibres and the tongue undergoes atrophy on the affected side. It is protruded toward the paralyzed side and may show fibrillary twitching.

The *symptoms* of involvement of one hypoglossal, either at its centre or in its course, are those of unilateral paralysis and atrophy of the tongue. When protruded, it is pushed toward the affected side, and there are fibrillary twitchings. The atrophy is usually marked and the mucous membrane on the affected side is thrown into folds. Articulation is not much impaired in the unilateral affection. There is a remarkable triad of symptoms, to which Hughlings Jackson first called attention—unilateral hemi-atrophy of the tongue, loss of power in the palate muscle, with paralysis of the larynx on the same side. When the disease is bilateral, the tongue lies almost motionless in the floor of the mouth; it is atrophied, and can not be protruded. Speech and mastication are extremely difficult and deglutition may be impaired. If the seat of the disease is above the nuclei, there may be little or no wasting. The condition is seen in progressive bulbar paralysis and occasionally in progressive muscular atrophy.

The *diagnosis* is readily made and the situation of the lesion can usually be determined, since when supra-nuclear there is associated hemiplegia and no wasting of the muscles of the tongue. Nuclear disease is only occasionally unilateral; most commonly bilateral and part of a bulbar paralysis. It should be borne in mind that the fibres of the hypoglossal may be involved within the medulla after leaving their nuclei. In such a case there may be paralysis of the tongue on one side and paralysis of the limbs on the opposite side, and the tongue, when protruded, is pushed toward the sound side.

Spasm.—This rare affection may be unilateral or bilateral. It is most frequently a part of some other convulsive disorder, such as epilepsy, chorea, or spasm of the facial muscles. In some cases of stuttering, spasm of the tongue precedes the explosive utterance of the words. It may occur in hysteria, and is said to follow reflex irritation in the fifth nerve. The most remarkable cases are those of paroxysmal clonic spasm, in which the tongue is rapidly thrust in and out, as many as forty or fifty times a minute. The prognosis is usually good.

IV. DISEASES OF THE SPINAL NERVES

CERVICAL PLEXUS

Occipito-cervical Neuralgia.—This involves the nerve territory supplied by the occipitalis major and minor, and the auricularis magnus nerves. The pains are chiefly in the back of the head and neck and in the ear. The condition may follow cold and is sometimes associated with stiffness of the neck or torticollis. Unless connected with it there exists disease of the bones or unless it is due to pressure of tumors, the outlook is usually good. There are tender points midway between the mastoid process and the spine and just above the parietal eminence, and between the sterno-mastoid and the trapezius. The affection may be due to direct pressure in carrying heavy weights.

Affections of the Phrenic Nerve.—Paralysis may follow a lesion in the

anterior horns at the level of the third and fourth cervical nerves, or may be due to compression of the nerve by tumors or aneurism. More rarely paralysis results from neuritis, diphtheritic or saturnine.

When the diaphragm is paralyzed respiration is carried on by the intercostal and accessory muscles. When the patient is quiet and at rest little may be noticed, but the abdomen retracts in inspiration and is forced out in expiration. On exertion or even on attempting to move there may be dyspnœa. If the paralysis sets in suddenly there may be dyspnœa and lividity, which is usually temporary (W. Pasteur). Intercurrent attacks of bronchitis seriously aggravate the condition. Difficulty in coughing, owing to the impossibility of drawing a full breath, adds greatly to the danger of this complication.

When the phrenic nerve is paralyzed on one side the paralysis may be scarcely noticeable, but careful inspection shows that the descent of the diaphragm is much less on the affected side.

The *diagnosis* of paralysis is not always easy, particularly in women, who habitually use this muscle less than men, and in whom the diaphragmatic breathing is less conspicuous. Immobility of the diaphragm is not uncommon, particularly in diaphragmatic pleurisy, in large effusions, and in extensive emphysema. The muscle itself may be degenerated and its power impaired.

Owing to the lessened action of the diaphragm, there is a tendency to accumulation of blood at the bases of the lungs, and there may be impaired resonance and signs of œdema. As a rule, however, the paralysis is not confined to this muscle, but is part of a general neuritis or an anterior poliomyelitis, and there are other symptoms of value in determining its presence. The outlook is usually serious. Pasteur states that of 15 cases following diphtheria only 8 recovered. The treatment is that of the neuritis or poliomyelitis.

Hiccough.—Here may, perhaps, best be considered this remarkable symptom, caused by intermittent, sudden contraction of the diaphragm. The mechanism, however, is complex, and while the afferent impressions to the respiratory centre may be peripheral or central the efferent are distributed through the phrenic nerve to the diaphragm, causing the intermittent spasm, and through the laryngeal branches of the vagus to the glottis, causing sudden closure as the air is rapidly inspired. There are various groups:

(*a*) Inflammatory, seen particularly in affections of the abdominal viscera, gastritis, peritonitis, hernia, internal strangulation, appendicitis, suppurative pancreatitis, and in the severe forms of typhoid fever.

(*b*) Irritative, as in the direct stimulation of the diaphragm when very hot substances are swallowed, in disease of the œsophagus near the diaphragm, and in many conditions of gastric and intestinal disorder, more particularly those associated with flatus.

(*c*) Specific, or, perhaps more properly, *idiopathic*, in which no evident causes are present. In these cases there is usually some constitutional taint, as gout, diabetes, or chronic Bright's disease. I have seen several instances of obstinate hiccough in the later stages of chronic interstitial nephritis.

(*d*) Neurotic, cases in which the primary cause is in the nervous system; hysteria, epilepsy, shock, or cerebral tumors. Of these cases the hysterical are, perhaps, the most obstinate.

Museum p 200 – Bilateral ab. Nerve common on left side,
many cells of development muscles. Small hypoplasia of
of motor fibres in nerve roots.
Symptoms un. nach Bildbet. 15 F 30.

The TREATMENT is often very unsatisfactory. Sometimes in the milder forms a sudden reflex irritation will check it at once. Readers of Plato's Symposium will remember that the physician Eryximachus recommended to Aristophanes, who had hiccough from eating too much, either to hold his breath (which for trivial forms of hiccough is very satisfactory) or to gargle with a little water; but if it still continued, "tickle your nose with something and sneeze; and if you sneeze once or twice even the most violent hiccough is sure to go." The attack must have been of some severity, as it is stated subsequently that the hiccough did not disappear until Aristophanes had resorted to the sneezing.

Ice, a teaspoonful of salt and lemon juice, or salt and vinegar, or a teaspoonful of raw spirits may be tried. When the hiccough is due to gastric irritation, lavage is sometimes promptly curative. I saw a case of a week's duration cured by a hypodermic injection of gr. ⅛ (0.008 gm.) of apomorphia. In obstinate cases the various antispasmodics have been used in succession. Pilocarpine has been recommended. The ether spray on the epigastrium may be promptly curative. Hypodermics of morphia, inhalations of chloroform, the use of nitrite of amyl and of nitroglycerin have been beneficial in some cases. Galvanism over the phrenic nerve, or pressure on the nerves, applied between the heads of the sterno-cleido-mastoid muscles may be used. Strong traction upon the tongue may give immediate relief. Of all measures morphia used freely is the best.

BRACHIAL PLEXUS

Cervical Rib Pressure.—Symptoms from pressure of this anomaly are by no means rare. It is usually bilateral, but the rib may be much longer on one side than on the other. The rib may be short and straight with the subclavian artery and brachial plexus in front of it, or longer and curved with the subclavian artery on its upper surface in a groove, in which case the artery is lengthened and elevated in the neck. Only about 5 or 10 per cent. of the cases have any trouble. The symptoms are either from pressure on the artery or on the nerve. The pulsation of the abnormally high artery may be mistaken for aneurism. In a few rare instances aneurism has occurred at the site of the pressure, and there have been cases of thrombosis with gangrene of the finger tips. I have seen three cases in which on exertion the arm became swollen, red and hot with numbness and tingling, but when quiet and at rest there was no inconvenience.

Affections of the nerves are more frequent and important. Pain is common, corresponding, as a rule, to the distribution of the eighth cervical and first dorsal roots, extending along the ulnar border of the forearm to the wrist or fingers. In other cases there is marked pressure on the brachial plexus with partial paralysis and wasting of the intrinsic muscles of the hand. There may be anæsthesia or hyperæsthesia of the inner aspect of the arm and the ulnar half of the hand. In a few instances there has been pressure on the cervical sympathetic nerve.

The condition is, as a rule, readily recognized, sometimes by palpation, always with the X-ray picture.

Combined Paralysis.—The plexus may be involved in the supraclavicular region by compression of the nerve trunks as they leave the spine, or by tumors and other morbid processes in the neck. Below the clavicle lesions are more common and result from injuries following dislocation or fracture, sometimes from neuritis. A cervical rib may lead to a pressure paralysis of the lower cord of the plexus. A not infrequent form of injury in this region follows falls or blows on the neck, which by lateral flexion of the head and depression of the shoulder seriously stretch the plexus. The entire plexus may be ruptured and the arm be totally paralyzed. The rupture may occur anywhere between the vertebræ and the clavicle, and involve all the cords of the plexus, or only the upper ones. The so-called "obstetrical palsy," due to drawing apart of the head and the shoulder during delivery, is an instance of this sort of injury. In these cases, however, the rupture of the plexus is usually only a partial one, involving its upper cord alone, so that the deltoid, biceps, supra- and infra-spinati, brachialis anticus, and supinator longus muscles may alone be affected. When the entire plexus has been ruptured a complete motor and sensory paralysis of the arm is produced. The roots may even be torn away from the spinal cord. The pupil will then be contracted on the side of the injury, and the arm hang from the body like a flail. Another common cause of lesion of the brachial plexus is luxation of the head of the humerus, particularly the subcoracoid form.

A primary neuritis of the brachial plexus is rare. More commonly the process is an ascending neuritis from a lesion of a peripheral branch, involving first the radial or ulnar nerves, and spreading upward to the plexus, producing gradually complete loss of power in the arm.

Lesions of Individual Nerves of the Plexus.—(*a*) Long Thoracic Nerve.—*Serratus paralysis* follows injury to this nerve in the neck, usually by direct pressure in carrying loads, and is very common in soldiers. It may be due to a neuritis following an acute infection or exposure. Isolated serratus paralysis is rare. It usually occurs in connection with paralysis of other muscles of the shoulder girdle, as in the myopathies and in progressive muscular atrophy. Concomitant trapezius paralysis is the most frequent. In the isolated paralysis there is little or no deformity with the hands hanging by the sides. There are slight abnormal obliquity of the posterior border of the scapula and prominence of the inferior angle, but when, as so commonly happens, the middle part of the trapezius is also paralyzed the deformity is marked. The shoulder is at a lower level, the inferior angle of the scapula is displaced inward and upward, and the superior angle projects upward. When the arms are held out in front at right angles to the body the scapula becomes winged and stands out prominently. The arm can not, as a rule, be raised above the horizontal. The outlook of the cases due to injury or to neuritis is good.

(*b*) Circumflex Nerve.—This supplies the deltoid and the teres minor. The nerve is apt to be involved in injuries, in dislocations, bruising by a crutch, or sometimes by extension of inflammation from the joint. Occasionally the paralysis arises from a pressure neuritis during an illness. As a consequence of loss of power in the deltoid, the arm can not be raised. The wasting is usually marked and changes the shape of the shoulder. Sensation may also be impaired in the skin over the muscle. The joint may be relaxed and

there may be a distinct space between the head of the humerus and the acromion.

(*c*) Musculo-spiral Paralysis; Radial Paralysis.—This is one of the most common of peripheral palsies, and results from the exposed position of the musculo-spiral nerve. It is often bruised in the use of the crutch, by injuries of the arm, blows, or fractures. It is frequently injured when a person falls asleep with the arm over the back of a chair, or by pressure of the body upon the arm when a person is sleeping on a bench or on the ground. It may be paralyzed by sudden violent contraction of the triceps. It is sometimes involved in a neuritis from cold, but this is uncommon in comparison with other causes. The paralysis of lead poisoning is the result of involvement of certain branches of this nerve.

A lesion when high up involves the triceps, the brachialis anticus, and the supinator longus, as well as the extensors of the wrist and fingers. Naturally, in lesions just above the elbow the arm muscles and the supinator longus are spared. The most characteristic feature of the paralysis is the wrist-drop and the inability to extend the first phalanges of the fingers and thumb. In the pressure palsies the supinators are usually involved and the movements of supination can not be accomplished. The sensations may be impaired, or there may be marked tingling, but the loss of sensation is rarely so pronounced as that of motion.

The affection is readily recognized, but it is sometimes difficult to say upon what it depends. The sleep and pressure palsies are, as a rule, unilateral and involve the supinator longus. The paralysis from lead is bilateral and the supinators are unaffected. Bilateral wrist-drop is a very common symptom in many forms of multiple neuritis, particularly the alcoholic; but the mode of onset and the involvement of the legs and arms are features which make the diagnosis easy. The duration and course of the musculo-spiral paralyses are very variable. The pressure palsies may disappear in a few days. Recovery is the rule, even when the affection lasts for many weeks. The electrical examination is of importance in the prognosis, and the rules laid down under paralysis of the facial nerve hold good here.

The treatment is that of neuritis.

(*d*) Ulnar Nerve.—The motor branches supply the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, the adductor and the inner head of the short flexor of the thumb, and the ulnar flexor of the wrist. The sensory branches supply the ulnar side of the hand—two and a half fingers on the back, and one and a half fingers on the front. Paralysis may result from pressure, usually at the elbow joint, although the nerve is here protected. Possibly the neuritis in the ulnar nerve in some cases of acute illness may be due to this cause. Gowers mentions the case of a lady who twice had ulnar neuritis after confinement. Owing to paralysis of the ulnar flexor of the wrist, the hand moves toward the radial side; adduction of the thumb is impossible; the first phalanges can not be flexed, and the others can not be extended. In long standing cases the first phalanges are overextended and the others strongly flexed, producing the claw-hand; but this is not so marked as in the progressive muscular atrophy. The loss of sensation corresponds to the sensory distribution just mentioned.

(*e*) Median Nerve.—This supplies the flexors of the fingers except the

ulnar half of the deep flexors, the abductor and the flexors of the thumb, the two radial lumbricales, the pronators, and the radial flexor of the wrist. The sensory fibres supply the radial side of the palm and the front of the thumb, the first two fingers and half the third finger, and the dorsal surfaces of the same three fingers.

This nerve is seldom involved alone. Paralysis results from injury and occasionally from neuritis. The signs are inability to pronate the forearm beyond the mid-position. The wrist can be flexed only toward the ulnar side; the thumb can not be opposed to the tips of fingers. The second phalanges can not be flexed on the first; the distal phalanges of the first and second fingers can not be flexed; but in the third and fourth fingers this action can be performed by the ulnar half of the flexor profundus. The loss of sensation is in the region corresponding to the sensory distribution already mentioned. The wasting of the thumb muscles, which is usually marked in this paralysis, gives to it a characteristic appearance.

LUMBAR AND SACRAL PLEXUSES

Lumbar Plexus.—The lumbar plexus is sometimes involved in growths of the lymph glands, in psoas abscess, and in disease of the bones of the vertebræ. When paralyzed the *obturator nerve* is occasionally injured during parturition. When paralyzed the power is lost over the abductors of the thigh and one leg can not be crossed over the other. Outward rotation is also disturbed. The *anterior crural nerve* is sometimes involved in wounds or in dislocation of the hip-joint, less commonly during parturition, and sometimes by disease of the bones and in psoas abscess. The special symptoms of affection of this nerve are paralysis of the extensors of the knee with wasting of the muscles, anæsthesia of the antero-lateral parts of the thigh and of the inner side of the leg to the big toe. This nerve is sometimes involved early in growths about the spine, and there may be pain in its area of distribution. Loss of the power of abducting the thigh results from paralysis of the *gluteal nerve*, which is distributed to the gluteus medius and minimus muscles.

External Cutaneous Nerve.—A peculiar form of sensory disturbance, confined to the territory of this nerve, was first described by Bernhardt in 1895, and a few months later by Roth, who gave it the name of *meralgia paræsthetica*. The disease is probably due to a neuritis which seems to originate in that part of the nerve where it passes under Poupart's ligament, just internal to the anterior superior iliac spine. The nerve is usually tender on pressure at this point. The disease is more common in men. Musser and Sailer in 1900 collected 99 cases, of which 75 were in men. A large number of the cases are attributable to direct traumatism or to simple pressure on the nerve by the aponeurotic canal through which it passes. Pregnancy is among the more common causes in women. The sensory disturbances consist of various forms of paræsthesia located over the outer side of the thigh, oftentimes with some actual diminution in the acuity of sense perception. The symptoms in varying intensity may persist for years, and the discomfort in some cases be so great, and so much exaggerated even by the mere touch of the clothing, that patients may be greatly incapacitated thereby. Excision of the nerve as it passes under Poupart's ligament has given good results.

Anosmia sensorii occurs. Deficit in hydropathia sensibilis (by
affection [illegible] with no motile nerve branches) is unexplained.
Sensibilis [illegible] over area becomes illicit. Obtuse from alcoholic & hair
follicles
phen. of cutis anosmica is lost.
Perverted sens. am present. - Shutting sensibility pains.
Tr. nutzos - dendritic pain below - (often not mentioned)

idiopathic [illegible] and attack

pericarditis [illegible] inflamm. cardiac sheath – not purul-
always a specific cause at origin of nerve– [illegible] specific joint cond

Sacral Plexus.—The sacral plexus is frequently involved in tumors and inflammations within the pelvis and may be injured during parturition. Neuritis is common, usually an extension from the sciatic nerve.

Goldthwaite calls attention to the fact that the lumbo-sacral articulation varies very greatly in its stability, and actual displacement of the bones may result with separation of the posterior portion of the intervertebral disc. The cauda equina, or the nerve roots, may be compressed. With displacement on one side the spine is rotated and the articular process of the fifth is drawn into the spinal canal, with such narrowing that paraplegia may result, and he reports a remarkable case in which the paralysis came on during the application of a plaster jacket. Weakness of the joints or the partial displacements may cause irritation of the nerves inside and outside the canal with resulting bilateral sciatica.

Of the branches, the *sciatic nerve,* when injured at or near the notch, causes paralysis of the flexors of the legs and the muscles below the knee, but injury below the middle of the thigh involves only the latter muscles. There is also anæsthesia of the outer half of the leg, the sole, and the greater portion of the dorsum of the foot. Wasting of the muscles frequently follows, and there may be trophic disturbances. In paralysis of one sciatic the leg is fixed at the knee by the action of the quadriceps extensor and the patient is able to walk.

Paralysis of the *small sciatic nerve* is rarely seen. The gluteus maximus is involved and there may be difficulty in rising from a seat. There is a strip of anæsthesia along the back of the middle third of the thigh.

External Popliteal Nerve.—Paralysis involves the peronæi, the long extensor of the toes, tibialis anticus, and the extensor brevis digitorum. The ankle can not be flexed, resulting in a condition known as foot-drop, and as the toes can not be raised the whole leg must be lifted, producing the characteristic *steppage* gait seen in so many forms of peripheral neuritis. In long-standing cases the foot is permanently extended and there is wasting of the anterior tibial and peroneal muscles. The loss of sensation is in the outer half of the front of the leg and on the dorsum of the foot.

Internal Popliteal Nerve.—When paralyzed, plantar flexion of the foot and flexion of the toes are impossible. The foot can not be adducted, nor can the patient rise on tiptoe. In long standing cases talipes calcaneus follows and the toes assume a claw-like position from secondary contracture, due to over-extension of the proximal and flexion of the second and third phalanges.

SCIATICA

This is, as a rule, a neuritis either of the sciatic nerve or of its cords of origin. It may in some instances be a functional neurosis or neuralgia.

It occurs most commonly in adult males. A history of rheumatism or of gout is present in many cases. Exposure to cold, particularly after heavy muscular exertion, or a severe wetting are not uncommon causes. Within the pelvis the nerves may be compressed by large ovarian or uterine tumors, by lymphadenomata, by the fetal head during labor; occasionally lesions of the hip-joint induce a secondary sciatica. More commonly, however, the condition is due to chronic arthritis of the spinal column. The condition of the nerve

has been examined in a few cases, and it has often been seen in the operation of stretching. It is, as a rule, swollen, reddened, and in a condition of interstitial neuritis. The affection may be most intense at the sciatic notch or in the nerve about the middle of the thigh.

Symptoms.—Of the symptoms, pain is the most constant and troublesome. The onset may be severe, with slight pyrexia, but, as a rule, it is gradual, and for a time there is only slight pain in the back of the thigh, particularly in certain positions or after exertion. Soon the pain becomes more intense and, instead of being limited to the upper portion of the nerve, extends down the thigh, reaching the foot and radiating over the entire distribution of the nerve. The patient can often point out the most sensitive spots, usually at the notch or in the middle of the thigh; and on pressure these are exquisitely painful. The pain is described as gnawing or burning, and is usually constant, but in some instances is paroxysmal, and often worse at night. On walking it may be very great; the knee is bent and the patient treads on the toes, so as to relieve the tension on the nerve. In protracted cases there may be much wasting of the muscles, but the reaction of degeneration can seldom be obtained. In these chronic cases cramp may occur and fibrillar contractions. Herpes may develop, but this is unusual. In rare instances the neuritis ascends and involves the spinal cord.

Duration and Course.—The duration and course are extremely variable. As a rule, it is an obstinate affection, lasting for months, or even, with slight remissions, for years. Relapses are not uncommon, and the disease may be relieved in one nerve only to appear in the other. In the severer forms the patient is bedridden, and such cases prove among the most distressing and trying which the physician is called upon to treat.

Diagnosis.—In the diagnosis it is important, in the first place, to determine whether the disease is primary, or secondary to some affection of the pelvis or of the spinal cord. A careful rectal examination should be made, and, in women, pelvic tumor should be excluded. Lumbago may be confounded with it. Affections of the hip-joint are easily distinguished by the absence of tenderness in the course of the nerve and the sense of pain on movement of the hip-joint or on pressure in the region of the trochanter. There are instances of sacro-iliac disease in which the patient complains of pain in the upper part of the thigh, which may sometimes radiate; but careful examination will readily distinguish between the affections. Pressure on the nerve trunks of the cauda equina, as a rule, causes bilateral pain and disturbances of sensation, and, as double sciatica is rare, these circumstances always suggest lesion of the nerve roots. Between the severe lightning pains of tabes and sciatica the differences are usually well defined. It is not to be forgotten that in a certain number of cases of so-called rheumatic sciatica the condition is a myositis, or, as Gowers calls it, a fibrositis. There is no tenderness along the course of the sciatic nerve, but there is pain in the gluteal region, with disability and Lasègue's sign, i. e., inability to extend the leg completely when the thigh is flexed on the abdomen.

Treatment.—The spinal column should be carefully and systematically examined, for numerous cases have been relieved by orthopædic procedures. The pelvic organs should also be investigated. Constitutional conditions, such as rheumatism and gout, should receive appropriate treatment. In a few cases

Pain when [illegible] is [illegible] of [illegible]

but

diseases elsewhere. The lower spine, and sacro-iliac region & hip joint should be carefully examined. If any one but lung lesion is found carefully, should be made of its cause (focus of infection, chiari &c.)

scheme or Duke's sacro-iliac. Disease.

pain. rest & [illegible] stretch [illegible] the nerve. - pain. Duke's sacro-il. jt. Disease straight leg [illegible] pain on [illegible] - [illegible]

Tub. of 5 lumbar - caries of sciatica

arthritis of spine. " "

5 lumbar vert. spin changes from normal

e.g. long transv. process

Rogers. J.A.M.A. - Feb 10. 17 50 cases

4 q. lesion of lower spine. lumbo-sac. region - il. j/t

1 sarcoma of pituitary

17 were sacro-iliac.

21 " chronic "

8 had vert. arthritis

with pronounced rheumatic history, which come on acutely with fever, the salicylates seem to do good. In other instances they are quite useless. If there is a suspicion of syphilis, the iodide of potassium should be employed, and in gouty cases salines.

Rest in bed with fixation of the limb by means of a long splint is a most valuable method of treatment in many cases, one upon which Weir Mitchell has specially insisted. I have known it to relieve, and in some instances to cure, obstinate and protracted cases which had resisted all other treatment. Hydrotherapy is sometimes satisfactory, particularly the warm baths or the mud baths. Many cases are relieved by a prolonged residence at one of the thermal springs. Antipyrin, antifebrin, and quinine are of doubtful benefit.

Local applications are more beneficial. The hot iron or the thermo-cautery or blisters relieve the pain temporarily. Deep injections into the nerves give great relief and may be necessary for the pain. It is best to use cocaine at first, in doses of from an eighth to a quarter of a grain (0.008 to 0.016 gm.). If the pain is unbearable morphia may be used, but it is a dangerous remedy in sciatica and should be withheld as long as possible. The disease is so protracted, so liable to relapse, and the patient's *morale* so undermined by the constant worry and the sleepless nights, that the danger of contracting the morphia habit is very great. On no consideration should the patient be permitted to use the hypodermic needle himself. It is remarkable how promptly, in some cases, the injection of distilled water into the nerve will relieve the pain. Acupuncture may also be tried; the needles should be thrust deeply into the most painful spot for a distance of about 2 inches, and left for from fifteen to twenty minutes. The injection of chloroform into the nerve has also been recommended.

Electricity is an uncertain remedy. Sometimes it gives prompt relief; in other cases it may be used for weeks without the slightest benefit. It is most serviceable in the chronic cases in which there is wasting of the legs, and should be combined with massage. The galvanic current should be used; a flat electrode should be placed over the sciatic notch, and a smaller one used along the course of the nerve and its branches. In very obstinate cases nerve-stretching may be employed. It is sometimes successful; but in other instances the condition recurs and is as bad as ever.

G. GENERAL AND FUNCTIONAL DISEASES

I. PARALYSIS AGITANS

(*Parkinson's Disease; Shaking Palsy*)

Definition.—A chronic affection of the nervous system, characterized by muscular weakness, tremors, and rigidity.

Etiology.—Men are more frequently affected than women. It rarely occurs under forty, but instances have been reported in which the disease began about the twentieth year. It is by no means an uncommon affection. Direct heredity is rare, but the patients often belong to families in which there are other nervous affections. Among exciting causes may be mentioned exposure

to cold and wet, and business worries and anxieties. In some instances the disease has followed directly upon severe mental shock or trauma. Cases have been described after the specific fevers. Malaria is believed by some to be an important factor, but of this there is no satisfactory evidence.

Morbid Anatomy.—No constant lesions have been found. The similarity between certain of the features of Parkinson's disease and those of old age suggests that the affection may depend upon a premature senility of certain regions of the brain. Our organs do not age uniformly, but in some, owing to hereditary disposition, the process may be more rapid than in others. "Parkinson's disease has no characteristic lesions, but, on the other hand, it is not a neurosis. It has for anatomical basis the lesions of cerebro-spinal senility, which only differ from those of true senility in their early onset and greater intensity" (Dubief). The important changes are doubtless in the cerebral cortex. No special changes have been found in the organs of internal secretion.

Symptoms.—The disease begins gradually, usually in one or other hand, and the tremor may be either constant or intermittent. With this may be associated weakness or stiffness. At first these symptoms may be present only after exertion. Although the onset is slow and gradual in nearly all cases, there are instances in which it sets in abruptly after fright or trauma. When well established the disease is very characteristic, and the diagnosis can be made at a glance. The four prominent symptoms are tremor, weakness, rigidity, and the attitude.

Tremor.—This may be in the four extremities or confined to hands or feet; the head is not so commonly affected. The tremor is usually marked in the hands, and the thumb and forefinger display the motion made in the act of rolling a pill. At the wrist there are movements of pronation and supination, and, though less marked, of flexion and extension. The upper-arm muscles are rarely involved. In the legs the movement is most evident at the ankle-joint, and less in the toes than in the fingers. Shaking of the head is less frequent, but does occur, and is usually vertical, not rotatory. The rate of oscillation is about five per second. Any emotion exaggerates the movement. The attempt at a voluntary movement may check the tremor (the patient may be able to thread a needle), but it returns with increased intensity. The tremors cease, as a rule, during sleep, but persist when the muscles are not in use. The writing of the patient is tremulous and zigzag.

Weakness.—Loss of power is present in all cases, and may occur even before the tremor, but is not very striking, as tested by the dynamometer, until the late stages. The weakness is greatest where the tremor is most developed. The movements, too, are remarkably slow. There is rarely complete loss of power.

Rigidity may early be expressed in a slowness and stiffness in the voluntary movements, which are performed with some effort and difficulty, and all the actions of the patient are deliberate. This rigidity is in all the muscles, and leads ultimately to the characteristic attitude.

Attitude and Gait.—The head is bent forward, the back is bowed, and the arms are held away from the body and are somewhat flexed at the elbow-joints. The face is expressionless, and the movements of the lips are slow.

The eyebrows are elevated, and the whole expression is immobile or mask-like, the so-called Parkinson's mask. The voice, as pointed out by Buzzard, is apt to be shrill and piping, and there is often a hesitancy in beginning a sentence; then the words are uttered with rapidity, as if the patient was in a hurry. This is sometimes in striking contrast to the scanning speech of insular sclerosis. The fingers are flexed and in the position assumed when the hand is at rest; in the late stages they can not be extended. Occasionally there is overextension of the terminal phalanges. The hand is usually turned toward the ulnar side and the attitude somewhat resembles that of advanced cases of rheumatoid arthritis. In the late stages there are contractures at the elbows, knees, and ankles. The movements of the patient are characterized by great deliberation. He rises from the chair slowly in the stooping attitude, with the head projecting forward. In attempting to walk the steps are short and hurried, and, as Trousseau remarks, he appears to be running after his centre of gravity. This is termed festination or propulsion, in contradistinction to a peculiar gait observed when the patient is pulled backward, when he makes a number of steps and would fall over if not prevented—retropulsion.

The REFLEXES are normal in most cases, but in a few they are exaggerated.

Of SENSORY disturbances Charcot has noted abnormal alterations in the temperature sense. The patient may complain of subjective sensations of heat, either general or local—a phenomenon which may be present on one side only and associated with an actual increase of the surface temperature, as much as 6° F. (Gowers). In other instances, patients complain of cold. Localized sweating may be present. The skin, especially of the forehead, may be thickened. The mental condition rarely shows any change.

VARIATIONS IN THE SYMPTOMS.—The tremor may be absent, but the rigidity, weakness, and attitude are sufficient to make the diagnosis. The disease may be hemiplegic in character, involving only one side or even one limb. Usually these are but stages of the disease.

Diagnosis.—In well developed cases the disease is recognized at a glance. The attitude, gait, stiffness, and mask-like expression are points of as much importance as the oscillations, and usually serve to separate the cases from senile and other forms of tremor. Disseminated sclerosis develops earlier, and is characterized by the nystagmus, and the scanning speech, and does not present the *attitude* so constant in paralysis agitans. Yet Schultze and Sachs have reported cases in which the signs of multiple sclerosis have been associated with those of paralysis. The hemiplegic form might be confounded with post-hemiplegic tremor, but the history, the mode of onset, and the greatly increased reflexes would be sufficient to distinguish the two. The Parkinsonian face is of great importance in the diagnosis of the obscure and anomalous forms.

The disease is incurable. Periods of improvement may occur, but the tendency is for the affection to proceed progressively downward. It is a slow, degenerative process and the cases last for years.

Treatment.—There is no method which can be recommended as satisfactory, in any respect. Arsenic, opium, hyoscine, and the extract of the parathyroid gland may be tried and sometimes give relief, but are not curative. The friends should be told frankly that the disease is incurable, and that

nothing can be done except to attend to the physical comforts of the patient. Regulated and systematized exercises should be carried out.

OTHER FORMS OF TREMOR

Simple Tremor.—This is occasionally found in persons in whom it is impossible to assign any cause. It may be transient or persist for an indefinite time. It is often extremely slight, and is aggravated by all causes which lower the vitality.

Hereditary Tremor.—C. L. Dana has reported remarkable cases of hereditary tremor. It occurred in all the members of one family, and beginning in infancy continued without producing any serious changes.

Senile Tremor.—With advancing age tremulousness during muscular movements is extremely common, but is rarely seen under seventy. It is always a fine tremor, which begins in the hands and often extends to the muscles of the neck, causing slight movement of the head.

Toxic tremor is seen chiefly as an effect of tobacco, alcohol, lead, or mercury; more rarely in arsenical or opium poisoning. In elderly men who smoke much it may be entirely due to the tobacco. One of the commonest forms of this is the alcoholic tremor, which occurs only on movement and has considerable range. Lead tremor is considered under lead poisoning, of which it constitutes a very important symptom.

Hysterical tremor, which usually occurs under circumstances which make the diagnosis easy, will be considered in the section on hysteria.

II. ACUTE CHOREA

(*Sydenham's Chorea; St. Vitus's Dance*)

Definition.—A disease chiefly affecting children, characterized by irregular, involuntary contraction of the muscles, a variable amount of psychical disturbance, and a remarkable liability to acute endocarditis.

Etiology.—Sex.—Of 554 cases ~~which I analyzed from~~ the Philadelphia Infirmary for Diseases of the Nervous System, 71 per cent. were in females and 29 per cent. in males. Of 808 Johns Hopkins Hospital cases, 71.2 per cent. were females (Thayer and Thomas).

Age.—The disease is most common between the ages of five and fifteen. Of 522 cases, 380 occurred in this period; 84.5 per cent. in Thayer and Thomas' series. It is rare among the negroes and native races of America. Only 25 of the Johns Hopkins Hospital cases were in negroes. ~~The cases are most numerous~~ when the mean relative humidity ~~is excessive and the barometric~~ pressure low (Lewis).

Rheumatism.—A casual relationship between rheumatism and chorea has been claimed by many since the time of Bright. The English and French writers maintain the closeness of this connection; on the other hand, German authors, as a rule, regard the connection as by no means very close. Of the 554 cases, in 15.5 per cent. there was a history of rheumatism in the family. In 88 cases, 15.8 per cent., there was a history of articular swelling, acute or

subacute. In 33 cases there were pains, sometimes described as rheumatic, in various parts, but not associated with joint trouble. Adding these to those with manifest articular trouble, the percentage is raised to nearly 21. It is rather remarkable that in our Baltimore series the percentage with a history of rheumatism was the same—21.6.

We find two groups of cases in which acute arthritis is present in chorea. In one, the arthritis antedates by some months or years the onset of the chorea, and does not recur before or during the attack. In the other group, the chorea sets in with or follows immediately upon the acute arthritis. In some instances it is impossible to decide whether the joint symptoms or the movements have appeared first. It is difficult to differentiate the cases of irregular pains without definite joint affection. It is probable that many of them are rheumatic, and yet I think it would be a mistake to regard as such all cases in children in which there are complaints of vague pains in the bones or muscles—so-called growing pains. It should never be forgotten, however, that a slight articular swelling may be the sole manifestation of rheumatic fever in a child—so slight, indeed, that the disease may be entirely overlooked.

Heart-disease.—Endocarditis is believed by some writers to be the cause of the disease. The particles of fibrin and vegetations from the valves pass as emboli to the cerebral vessels. On this view, which we shall discuss later, chorea is the result of an embolic process occurring in the course of a rheumatic endocarditis.

Infectious Diseases.—Scarlet fever with arthritic manifestations may be a direct antecedent. Sturges states that a history of previous whooping-cough occurs more frequently in choreic than in other children, but I find no evidence of this in the Infirmary records. With the exception of rheumatic fever, there is no intimate relationship between chorea and the acute diseases incident to childhood. It may be noted in contrast to this that the so-called canine chorea is a common sequel of distemper. Chorea has been known to develop in the course of an acute pyæmia, and to follow gonorrhœa and puerperal fever.

Anæmia is less often an antecedent than a sequence of chorea, and though cases develop in children who are anæmic and in poor health, this is by no means the rule. Chorea may develop in chlorotic girls at puberty.

Pregnancy.—A choreic patient may become pregnant; more frequently the disease occurs during pregnancy; sometimes it develops post partum. Buist, of Dundee, has tabulated carefully 226 cases: in 6 the chorea preceded the pregnancy; in 105 it occurred during the pregnancy; in 31 in recurrent pregnancies; 45 cases terminated fatally, and in 16 cases the chorea developed post partum. The alleged frequency in illegitimate primiparæ is not borne out by his figures. Beginning in the first three months were 108 cases, in the second three months 70 cases, in the last three months 25 cases. The disease is often severe, and maniacal symptoms may develop.

A tendency to the disease is found in certain families. In 80 cases there was a history of attacks of chorea in other members. In one instance both mother and grandmother had been affected. High-strung, excitable, nervous children are especially liable to the disease. *Fright* is considered a frequent cause, but in a large majority of the cases no close connection exists between the fright and the onset of the disease. Occasionally the attack sets in at

once. Mental worry, trouble, a sudden grief, or a scolding may apparently be the exciting cause. The strain of *education,* particularly in girls during the third hemidecade, is a most important factor in the etiology of the disease. Bright, intelligent, active minded girls from ten to fourteen, ambitious to do well at school, often stimulated in their efforts by teachers and parents, form a large contingent of the cases of chorea in hospital and private practice. Sturges has called special attention to this *school-made* chorea as one serious evil in our modern method of forced education. *Imitation,* which is mentioned as an exciting cause, is extremely rare, and does not appear to have influenced the onset in a single case in the Infirmary records.

The disease may rapidly follow an injury or a slight surgical operation. Reflex irritation was believed to play an important rôle in the disease, particularly the presence of worms or genital irritation; but I have met with no instance in which the disease could be attributed to either of these causes. Local spasm, particularly of the face—the habit chorea of Mitchell—may be associated with irritation in the nostrils and adenoid growths in the vault of the pharynx, as pointed out by Jacobi.

It has been claimed by Stevens that *ocular defects* lie at the basis of many cases of chorea, and that with the correction of these the irregular movements disappear. The investigations of De Schweinitz show that ocular defects do not occur in greater proportion in choreic than in other children. A majority of the cases in which operation has been followed by relief have been instances of *tic,* local or general.

Morbid Anatomy and Pathology.—No constant lesions have been found in the nervous system in acute chorea. Vascular changes, such as hyaline transformation, exudation of leucocytes, minute hæmorrhages, and thrombosis of the smaller arteries, have been described.

Embolism of the smaller cerebral vessels has been found, and there are on record 7 cases of embolism of the central artery of the retina (H. M. Thomas). Based on the presence of emboli, Kirkes and others have supported what is known as the embolic theory of the disease. Endocarditis is by far the most frequent lesion in Sydenham's chorea. With no disease, not excepting rheumatism, is it so constantly associated. I collected from the literature the records of 73 autopsies; there were 62 with endocarditis.* The endocarditis is usually of the simple variety, but the ulcerative form has occasionally been described.

We are still far from a solution of all the problems connected with chorea. Unfortunately, the word has been used to cover a series of totally diverse disorders of movement, so that there are still excellent observers who hold that chorea is only a symptom, and is not to be regarded as an etiological unit. The chorea of childhood, the disease which Sydenham described, presents, however, characteristics so unmistakable that it must be regarded as a definite, substantive affection. Some regard it as *a functional brain disorder* affecting the nerve centres controlling the motor apparatus, an instability of the nerve cells, brought about, one supposes by hyperæmia, another by anæmia, a third by psychical influences, a fourth by irritation, central or peripheral. Of the actual nature of this derangement we know nothing, nor, indeed, whether the changes

* Osler, "Chorea and Choreiform Affections."

are primary and the result of a faulty action of the cortical cells or whether the impulses are secondarily disturbed in their course down the motor path. The predominance of the disease in females, and its onset at a time when the education of the brain is rapidly developing, are etiological facts which Sturges has urged in favor of the view that chorea is an expression of functional instability of the nerve centres.

The *embolic theory* originally advanced by Kirkes has a solid basis of fact, but it is not comprehensive enough, as all of the cases can not be brought within its limits. There are instances without endocarditis and without, so far as can be ascertained, plugging of cerebral vessels; and there are also cases with extensive endocarditis in which the histological examination of the brain, so far as embolism is concerned, was negative. In favor of the embolic view is the experimental production in animals of chorea by Rosenthal, and later by Money, by injecting fine particles into the carotids.

Lately, as indeed might be expected, chorea has been regarded as an *infectious disease*. Nothing definite has yet been determined. In favor of this view it has been urged, as it is impossible to refer the chorea to endocarditis or the endocarditis in all cases to rheumatism, that both have their origin in a common cause, some infectious agent, which is capable also, in persons predisposed, of exciting articular disease. Cases have been reported in scarlet fever with arthritic manifestations, in puerperal fever, and rheumatism, also after gonorrhœa, and such facts are suggestive at least of the association of the disease with infective processes. Possibly, as has been suggested by some writers, the paralytic condition associated with chorea may be analogous to those which occur in typhoid and certain of the infectious diseases. On the other hand, there are conditions extremely difficult to harmonize with this view. The prominent psychical element is certainly one of the most serious objections, since there can be no doubt that ordinary chorea may rapidly follow a fright or a sudden emotion.

Symptoms.—Three groups of cases may be recognized—the mild, severe, and maniacal chorea.

Mild Chorea.—In this the affection of the muscles is slight, the speech is not seriously disturbed, and the general health not impaired. Premonitory symptoms are shown in restlessness and inability to sit still, a condition well characterized by the term "fidgets." There are emotional disturbances, such as crying spells, or sometimes night terrors. There may be pains in the limbs and headache. Digestive disturbances and anæmia may be present. A change in the temperament is frequently noticed, and a docile, quiet child may become cross and irritable. After these symptoms have persisted for a week or more the characteristic involuntary movements begin, and are often first noticed at the table, when the child spills a tumbler of water or upsets a plate. There may be only awkwardness or slight incoördination of voluntary movements, or constant irregular clonic spasms. The jerky, irregular character of the movements differentiates them from almost every other disorder of motion. In the mild cases only one hand, or the hand and face, are affected, and it may not spread to the other side.

In the second grade, the *severe form*, the movements become general and the patient may be unable to get about or to feed or undress herself, owing to the constant, irregular, clonic contractions of the various muscle groups.

The speech is also affected, and for days the child may not be able to talk. Often with the onset of the severer symptoms there is loss of power on one side or in the limb most affected.

The third and most extreme form, the so-called maniacal chorea, or *chorea insaniens*, is truly a terrible disease, and may develop out of the ordinary form. These cases are more common in adult women and may develop during pregnancy.

Chorea begins, as a rule, in the hands and arms, then involves the face, and subsequently the legs. The movements may be confined to one side—hemichorea. The attack begins oftenest on the right side, though occasionally it is general from the outset. One arm and the opposite leg may be involved. In nearly one-fourth of the cases speech is affected; this may amount only to an embarrassment or hesitancy, but in other instances it becomes an incoherent jumble. In very severe cases the child will make no attempt to speak. The inability is in articulation rather than in phonation. Paroxysms of panting and of hard expiration may occur, or odd sounds may be produced. As a rule the movements cease during sleep.

Paralysis.—A prominent symptom is muscular weakness, usually no more than a condition of paresis. The loss of power is slight, but the weakness may be shown by an enfeebled grip or by a dragging of the leg or limping. In his original account Sydenham refers to the "unsteady movements of one of the legs, which the patient drags." There may be extreme paresis with but few movements—the paralytic chorea of Todd. Occasionally a local paralysis or weakness remains after the attack.

Mutism is an interesting feature; for weeks the child may not say a word. It is more common in severe cases, but is not marked by special choreic unrest of the muscles of speech; it is probably a motor weakness. Complete recovery follows.

It is doubtful whether choreic spasms extend to the muscles of organic life. The rapid action and disturbed rhythm of the heart present nothing peculiar to the disease, and there is no support for the view that irregular contractions occur in the papillary muscles.

Heart Symptoms.—*Neurotic.*—As so many of the subjects of chorea are nervous girls, it is not surprising that a common symptom is a rapidly acting heart. Irregularity is not so special a feature in chorea as rapidity. The patients seldom complain of pain about the heart.

Hæmic Murmurs.—With anæmia and debility, not uncommon associates of chorea in the third or fourth week, we find a corresponding cardiac condition. The impulse is diffuse, perhaps wavy in thin children. The carotids throb visibly, and in the recumbent posture there may be pulsation in the cervical veins. On auscultation a systolic murmur is heard at the base, perhaps, too, at the apex, soft and blowing in quality.

Endocarditis.—As in rheumatism, so in chorea, acute valvulitis rarely gives evidence of its presence by symptoms. It must be sought, and clinical experience has shown that it is usually associated with murmurs at one or other of the cardiac orifices.

For the guidance of the practitioner these statements may be made:

(*a*) In thin, nervous children a systolic murmur of soft quality is extremely common at the base, with accentuation of the second sound, par-

ticularly at the second left costal cartilage, and is probably of no moment.

(*b*) A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

(*c*) A murmur of maximum intensity at the apex, with rough quality, and transmitted to the axilla or angle of the scapula, indicates an organic lesion of the mitral valve, and is usually associated with enlargement of the heart.

(*d*) When in doubt it is much safer to trust to the evidence of eye and hand than to that of the ear. If the apex beat is in the normal position, and the area of dulness not increased vertically or to the right of the sternum, there is probably no serious valvular disease.

(*e*) The endocarditis of chorea is almost invariably of the simple or warty form, and in itself is not dangerous; but it is apt to lead to those sclerotic changes in the valve which produce incompetency. Of 140 patients examined more than two years after the attack, I found the heart normal in 51; in 17 there was functional disturbance, and 72 presented signs of organic heart-disease.

(*f*) Pericarditis is an occasional complication of chorea, usually in cases with well-marked rheumatism.

In an analysis of the cases at the Johns Hopkins Hospital, Thayer found evidence of involvement of the heart in 25 per cent. of the out-patients and in more than 50 per cent. of the cases in the wards. Cardiac involvement was more common in the cases with a history of rheumatism, and was much more frequent in the relapses.

Sensory Disturbances.—Pain in the affected limbs is not common. Occasionally there is soreness on pressure. There are cases, usually of hemichorea, in which pain in the limbs is a marked symptom. Weir Mitchell has spoken of these as *painful choreas*. Tender points along the lines of emergence of the spinal nerves or along the course of the nerves of the limbs are rare.

Psychical disturbances are common, though in a majority of the cases slight in degree. Irritability of temper, marked wilfulness, and emotional outbreaks may indicate a complete change in the character of the child. There is deficiency in the powers of concentration, the memory is enfeebled, and the aptitude for study is lost. Rarely there is progressive impairment of the intellect with termination in actual dementia. Acute melancholia has been described. Hallucinations of sight and hearing may occur. Patients may behave in an odd and strange manner and do all sorts of meaningless acts. By far the most serious manifestation of this character is the maniacal delirium, occasionally associated with the very severe cases—*chorea insaniens*. Usually the motor disturbance in these cases is aggravated, but it has been overlooked and patients have been sent to an asylum.

The psychical element in chorea is apt to be neglected by the practitioner. It is always a good plan to tell the parents that it is not the muscles alone of the child which are affected, but that the general irritability and change of disposition, so often found, really form part of the disease.

The condition of the reflexes in chorea is usually normal. Trophic lesions rarely occur in chorea unless, as some writers have done, we regard the joint troubles as arthropathies occurring in the course of a cerebro-spinal disease.

Fever, usually slight, was present in all but one of 110 cases treated in my wards (Thayer). H. A. Hare states that in monochorea the temperature on the affected side may be elevated; but this is not an invariable rule. Endocarditis may occur with little if any rise in temperature; but, on the other hand, with an acute arthritis, severe endocarditis or pericarditis, and in the cases of maniacal chorea, the fever may range from 102° to 104°.

Cutaneous Affections.—The pigmentation, which is not uncommon, is due to the arsenic. Herpes zoster occasionally occurs. Certain skin eruptions, usually regarded as rheumatic in character, are not uncommon. Erythema nodosum has been described and I have seen several cases with a purpuric urticaria. There may, indeed, be the more aggravated condition of rheumatic purpura, known as Schönlein's *peliosis rheumatica*. Subcutaneous fibrous nodules, which have been noted by English observers in many cases of chorea, associated with rheumatism, are extremely rare in the United States.

Duration and Termination.—From eight to ten weeks is the average duration of an attack of moderate severity. Chronic chorea rarely follows the minor disease which we have been considering. The cases described under this designation in children are usually instances of cerebral sclerosis or Friedreich's ataxia; but occasionally an attack which has come on in the ordinary way persists for months or years, and recovery ultimately takes place. A slight grade of chorea, particularly noticeable under excitement, may persist for months in nervous children.

The tendency of chorea to recur has been noticed by all writers since Sydenham first made the observation. Of 410 cases analyzed for this purpose, 240 had one attack, 110 had two attacks, 35 three attacks, 10 four attacks, 12 five attacks, and 3 six attacks. The recurrence is apt to be vernal.

Recovery is the rule in children. The statistics of out-patient departments are not favorable for determining the mortality. A reliable estimate is that of the Collective Investigation Committee of the British Medical Association, in which 9 deaths were reported among 439 cases, about 2 per cent.

The paralysis rarely persists. Mental dulness may be present for a time, but usually passes away; permanent impairment of the mind is an exceptional sequence.

Diagnosis.—There are few diseases which present more characteristic features, and in a majority of instances the nature of the trouble is recognized at a glance; but there are several affections in children which may simulate and be mistaken for it.

(*a*) *Multiple and diffuse cerebral sclerosis.* The cases are often mistaken for ordinary chorea, and have been described in the literature as *chorea spastica*. There are doubtless chronic changes in the cortex. As a rule, the movements are readily distinguishable from those of true chorea, but the simulation is sometimes very close; the onset in infancy, the impaired intelligence, increased reflexes and in some instances rigidity, and the chronic course of the disease separate them sharply from true chorea.

(*b*) *Friedreich's ataxia.* Cases of this well-characterized disease were formerly classed as chorea. The slow, irregular, incoördinate movements, the scoliosis, the scanning speech, the early talipes, the nystagmus, and the family character of the disease are points which should render the diagnosis easy.

(*c*) In rare cases the paralytic form of chorea may be mistaken for *polio-*

myelitis or, when both legs are affected, for paraplegia of spinal origin; but this can be the case only when the choreic movements are very slight.

(*d*) *Hysteria* may simulate chorea minor most closely, and unless there are other manifestations it may be impossible to make a diagnosis. Most commonly, however, the movements in the so-called hysterical chorea are rhythmic and differ entirely from those of ordinary chorea.

(*e*) As mentioned above, the *mental symptoms* in maniacal chorea may mask the true nature of the disease and patients have even been sent to the asylum.

Treatment.—Abnormally bright, active minded children belonging to families with pronounced neurotic taint should be carefully watched from the ages of eight to fifteen and not allowed to overtax their mental powers. So frequently in children of this class does the attack of chorea date from the worry and stress incident to school examinations that the competition for prizes or places should be emphatically forbidden.

The treatment of the attack consists largely in attention to hygienic measures, with which alone, in time, a majority of the cases recover. Parents should be told to scan gently the faults and waywardness of choreic children. The psychical element, strongly developed in so many cases, is best treated by quiet and seclusion. The child should be confined to bed in the recumbent posture, and mental as well as bodily quiet enjoined. In private practice this is often impossible, but with well-to-do patients the disease is always serious enough to demand the assistance of a skilled nurse. Toys and dolls should not be allowed at first, for the child should be kept amused without excitement. The rest allays the hyper-excitability and reduces to a minimum the possibility of damage to the valve segments should endocarditis exist. Time and again have I seen very severe cases which had resisted treatment for weeks outside a hospital become quite, and the movements subside after two or three days of absolute rest in bed.

The child should be kept apart from other children and, if possible, from other members of the family, and should see only those persons directly concerned with the nursing of the case. In the latter period of the disease daily rubbings may be resorted to with great benefit.

The medical treatment of the disease is unsatisfactory; with the exception of arsenic, no remedy seems to have any influence in controlling the progress of the affection. Without any specific action, it certainly does good in many cases, probably by improving the general nutrition. It is conveniently given in the form of Fowler's solution, and the good effects are rarely seen until maximum doses are taken. It may be given as Martin originally advised (1813); he began "with five drops and increased one drop every day, until it might begin to disagree with the stomach or bowels." When the dose of 15 minims is reached, it may be continued for a week, and then again increased, if necessary, every day or two, until physiological effects are manifest. On the occurrence of these the drug should be stopped for three or four days. The practice of resuming the administration with smaller doses is rarely necessary, as tolerance is usually established and we can begin with the dose which the child was taking when the symptoms of saturation occurred. I have frequently given as much as 25 minims three times a day. Usually the signs of saturation are trivial but plain, but in very rare instances more serious symp-

toms develop. A fatal arsenical neuritis followed in the case of a child, aged eight, who took seven drops of Fowler's solution three times a day for ten days, then stopped for a week, and then took seven drops three times a day for fourteen days (Cary Gamble).

Of other medicines sedatives are useful in the severe attacks. Chloral is the most useful and may be begun in doses of five grains (0.3 gm.), gradually increased if necessary. Sodium bromide in the same dosage may be added. Belladonna has been found useful in some cases.

For its tonic effect electricity is sometimes useful; but it is not necessary as a routine treatment. The question of gymnastics is an important one. Early in the disease, when the movements are active, they are not advisable; but during convalescence carefully graduated exercises are undoubtedly beneficial. It is not well, however, to send a choreic child to a school gymnasium, as the stimulus of the other children and the excitement of the romping, violent play are very prejudicial.

Other points in treatment may be mentioned. Food should be simple and some children do best on a milk diet, the amount being rapidly increased. It is important to regulate the bowels and to attend carefully to the digestive functions. For the anæmia so often present preparations of iron are indicated.

In the severe cases with incessant movements, sleeplessness, dry tongue, and delirium, the important indication is to procure rest, for which purpose chloral may be freely given, and, if necessary, morphia. Chloroform inhalations may be necessary to control the intensity of the paroxysms, but the high rate of mortality in this class of cases illustrates how often our best endeavors are fruitless. The wet pack is sometimes very soothing and should be tried. As these patients are apt to sink rapidly into a low typhoid state with heart weakness, a supporting treatment is required from the outset.

Cases are found now and then which drag on from month to month without getting either better or worse and resist all modes of treatment. In such cases a combination of suggestion and passive movements, followed by voluntary movements under control, and later simple exercises, may be useful. Change of air and scene is sometimes followed by rapid improvement, and in these cases the treatment by rest and seclusion should always be given a full trial.

In all cases care should be taken to examine the nostrils, and glaring ocular defects should be properly corrected either by glasses or, if necessary, by operation.

After the child has recovered from the attack, the parents should be warned that return of the disease is by no means infrequent, and is particularly liable to follow overwork at school or debilitating influences of any kind. These relapses are apt to occur in the spring. Sydenham advised purging in order to prevent the vernal recurrence of the disease.

III. OTHER AFFECTIONS DESCRIBED AS CHOREA

Chorea Major: Pandemic Chorea.—The common name, St. Vitus's dance, applied to chorea has come to us from the middle ages, when under the influence of religious fervor there were epidemics characterized by great excitement,

gesticulations, and dancing. For the relief of these symptoms, when excessive, pilgrimages were made, and, in the Rhenish provinces, particularly to the Chapel of St. Vitus in Zebern. Epidemics of this sort occurred also during the nineteenth century, and descriptions of them among the early settlers in Kentucky have been given by Robertson and Yandell. It was unfortunate that Sydenham applied the term chorea to an affection in children totally distinct from this chorea major, which is in reality an hysterical manifestation under the influence of religious excitement.

Habit Spasm (*Habit Chorea*); **Convulsive Tic** (of the French).—Two groups of cases may be recognized under the designation of habit spasm—one in which there are simply localized spasmodic movements, and the other in which, in addition to this, there are explosive utterances and psychical symptoms, a condition to which French writers have given the name *tic convulsif*.

(*a*) Habit Spasm.—This is found chiefly in childhood, most frequently in girls from seven to fourteen years of age (Mitchell). In its simplest form there is a sudden, quick contraction of certain of the facial muscles, such as rapid winking or drawing of the mouth to one side, or the neck muscles are involved and there are unilateral movements of the head. The head is given a sudden, quick shake, and at the same time the eyes wink. A not infrequent form is the shrugging of one shoulder. The grimace or movement is repeated at irregular intervals, and is much aggravated by emotion. A short inspiratory sniff is not an uncommon symptom. The cases are found most frequently in children who are "out of sorts," or who have been growing rapidly, or who have inherited a tendency to neurotic disorders. Allied to or associated with this are some of the curious tricks of children. A boy ~~at my clinic~~ was in the habit every few moments of putting the middle finger into the mouth, biting it, and at the same time pressing his nose with the forefinger. Hartley Coleridge is said to have had a somewhat similar trick, only he bit his arm. In all these cases the habits of the child should be examined carefully, the nose and vault of the pharynx thoroughly inspected, and the eyes accurately tested. As a rule the condition is transient, and after persisting for a few months or longer gradually disappears. Occasionally a local spasm persists—twitching of the eyelids, or the facial grimace.

(*b*) Impulsive Tic (Gilles de la Tourette's Disease).—This remarkable affection, often mistaken for chorea, more frequently for habit spasm, is really a psychosis allied to hysteria, though in certain of its aspects it has the features of monomania. The disease begins, as a rule, in young children, occurring as early as the sixth year, though it may ~~develop~~ after puberty. There is usually a markedly neurotic family history. The special features ~~of the complaint~~ are:

(1) Involuntary muscular movements, usually affecting the facial or brachial muscles, but in aggravated cases all the muscles of the body may be involved and the movements may be extremely irregular and violent.

(2) Explosive utterances, which may resemble a bark or an inarticulate cry. A word heard may be mimicked at once and repeated over and over again, usually with the involuntary movements. To this the term *echolalia* has been applied. A much more distressing disturbance in these cases is *coprolalia*, or the use of bad language. A child of eight or ten may shock its

mother and friends by constantly using the word *damn* when making the involuntary movements, or by uttering all sorts of obscene words. Occasionally actions are mimicked—*echokinesis.*

(3) Associated with some of these cases are curious mental disturbances; the patient becomes the subject of a form of obsession or a fixed idea. In other cases the fixed idea takes the form of the impulse to touch objects, or it is a fixed idea about words—onomatomania—or the patient may feel compelled to count a number of times before doing certain actions—arithmomania.

The disease is well marked and readily distinguished from ordinary chorea. The movements have a larger range and are explosive in character. Tourette regards the coprolalia as the most distinctive feature of the disease. The prognosis is doubtful, I have, however, known recovery to follow.

Saltatory Spasm (*Latah; Myriachit; Jumpers*).—Bamberger has described a disease in which when the patient attempted to stand there were strong contractions in the leg muscles, which caused a jumping or springing motion. This occurs only when the patient attempts to stand. The affection has occurred in both men and women, more frequently in the former, and the subjects have usually shown marked neurotic tendencies. In many cases the condition has been transitory; in others it has persisted for years. Remarkable affections similar to this in certain points occur as a sort of epidemic neurosis. One of the most striking of these occurs among the "jumping Frenchmen" of Maine and Canada. As described by Beard and Thornton, the subjects are liable on any sudden emotion to jump violently and utter a loud cry or sound, and will obey any command or imitate any action without regard to its nature. The condition of echolalia is present in a marked degree. The "jumping" prevails in certain families.

A very similar disease prevails in parts of Russia and in Java and Borneo, where it is known by the names of myriachit and latah, the chief feature of which is mimicry by the patient of everything he sees or hears.

Chronic Chorea (*Huntington's Chorea*).—This is an affection characterized by irregular movements, disturbance of speech, and gradual dementia. It is frequently hereditary. Irving W. Lyon described it in 1863 as *chronic hereditary chorea* and traced the disease through five generations. Huntington, of Pomeroy, Ohio, at the time a practitioner on Long Island, gave, in 1872, in three brief paragraphs the salient points in connection with the disease—namely, the hereditary nature, the association with psychical troubles, and the late onset—between the thirtieth and fortieth years. The disease is not uncommon in the United States. Under the term chronic chorea may be grouped the hereditary form and the cases which come on without family disposition, either at middle life or, more commonly, in the aged—senile chorea. It is doubtful whether the cases in children with chronic choreiform movements, often with mental weakness and spastic condition of the legs, should go into this category.

The hereditary character of the disease is very striking; it has been traced through four or five generations. Huntington's father and grandfather, also physicians, had treated the disease in the family which he described. Osborn, of East Hampton, tells me that the disease still continues to recur in certain families described by Huntington, as it has done, so it is said, for fully two

centuries. An identical affection occurs without any hereditary disposition. The age of onset is late, rarely before the thirtieth or the thirty-fifth year.

The symptoms are very characteristic. The irregular movements are usually first seen in the hands, and the patient has slight difficulty in performing delicate manipulations or in writing. When well established the movements are disorderly, irregular, incoördinate rather than choreic, and have not the sharp, brusque motion of Sydenham's chorea. In the face there are slow, involuntary grimaces. In a well-developed case the gait is irregular, swaying, and somewhat like that of a drunken man. The speech is slow and difficult the syllables are badly pronounced and indistinct, but not definitely staccato. The mental impairment leads finally to dementia. The anatomical condition is a chronic diffuse cortical encephalitis not unlike that in general paralysis.

Rhythmic or Hysterical Chorea.—This is readily recognized by the rhythmical character of the movements. It may affect the muscles of the abdomen, producing the salaam convulsion, or involve the sterno-mastoid, producing a rhythmical movement of the head, or the psoas, or any group of muscles. In its orderly rhythm it resembles the canine chorea.

IV. INFANTILE CONVULSIONS

(*Eclampsia*)

Convulsive seizures similar to those of epilepsy are not infrequent in children and in adults. The fit may indeed be identical with epilepsy, from which the condition differs in that when the cause is removed there is no tendency for the fits to recur. Occasionally, however, the convulsions in children continue and develop into true epilepsy.

Etiology.—A convulsion in a child may be due to many causes, all of which lead to an unstable condition of the nerve centres, permitting sudden, excessive, and temporary nervous discharges. The following are the most important of them:

(1) *Debility*, resulting usually from gastro-intestinal disturbance. Convulsions frequently supervene toward the close of an attack of entero-colitis and recur, sometimes proving fatal. The death-rate in children from eclampsia rises steadily with that of gastro-intestinal disorders (Morris J. Lewis).

(2) *Peripheral Irritation.*—Dentition alone is rarely a cause of convulsions, but is often one of several factors in a feeble, unhealthy infant. The greatest mortality from convulsions is during the first six months, before the teeth have really cut through the gums. Other irritative causes are the overloading of the stomach with indigestible food. It has been suggested that some of these cases are toxic, owing to the absorption of poisonous ptomaines. Worms, to which convulsions are so frequently attributed, probably have little influence. Among other sources possible are phimosis and otitis.

(3) *Rickets.*—The observation of Sir William Jenner upon the association of rickets and convulsions has been amply confirmed. The spasms may be laryngeal, the so-called child-crowing, which, though convulsive in nature, can scarcely be reckoned under eclampsia. The influence of this condition is more apparent in Europe than in the United States, although rickets is a com-

mon disease, particularly among the colored people. Spasms, local or general, in rickets are probably associated with the condition of debility and malnutrition and with cranio-tabes.

(4) *Fever.*—In young children the onset of the infectious diseases is frequently with convulsions, which often take the place of a chill in the adult. It is not known upon what they depend. Scarlet fever, measles, and pneumonia are most often preceded by convulsions.

(5) *Congestion of the Brain.*—That extreme engorgement of the blood-vessels may produce convulsions is shown by their occasional occurrence in severe whooping-cough, but their rarity in this disease really indicates how small a part mechanical congestion plays in the production of fits.

(6) *Severe convulsions* usher in or accompany many of the serious diseases of the nervous system in children. In more than 50 per cent. of the cases of infantile hemiplegia the affection follows severe convulsions. They less frequently precede a spinal paralysis. They occur with meningitis, tuberculous or simple, and with tumors and other lesions of the brain.

And, lastly, convulsions may occur immediately after birth and persist for weeks or months. In such instances there has probably been meningeal hæmorrhage or serious injury to the cortex.

The most important question is the relation of convulsions in children to true epilepsy. In Gowers' figures of 1,450 cases of epilepsy, the attacks began in 180 during the first three years of life. Of 460 cases of epilepsy in children which I have analyzed, in 187 the fits began within the first three years. Of the total list the greatest number, 74, was in the first year. In nearly all these instances there was no interruption in the convulsions. That convulsions in early infancy are necessarily followed by epilepsy in after life is certainly a mistake.

Symptoms.—The attack may come on suddenly without any warning; more commonly it is preceded by a stage of restlessness, accompanied by twitching and perhaps grinding of the teeth. It is rarely so complete in its stages as true epilepsy. The spasm begins usually in the hands, most commonly in the right hand. The eyes are fixed and staring or are rolled up. The body becomes stiff and breathing is suspended for a moment or two by tonic spasm of the respiratory muscles, in consequence of which the face becomes congested. Clonic convulsions follow, the eyes are rolled about, the hands and arms twitch, or are fixed and extended in rhythmical movements, the face is contorted, and the head is retracted. The attack gradually subsides and the child sleeps or passes into a state of stupor. Following indigestion the attack may be single, but in rickets and intestinal disorders it is apt to be repeated. Sometimes the attacks follow each other with great rapidity, so that the child never rouses but dies in a deep coma. If the convulsion has been limited chiefly to one side there may be slight paresis after recovery, or in instances in which the convulsions usher in infantile hemiplegia, when the child arouses, one side is completely paralyzed. During the fit the temperature is often raised. Death rarely occurs from the convulsion itself, except in debilitated children or when the attacks recur with great frequency. In the so-called hydrocephaloid state in connection with protracted diarrhœa convulsions may close the scene.

Diagnosis.—Coming on when the subject is in full health, the attack is

probably due either to an overloaded stomach, to some peripheral irritation, or occasionally to trauma. Setting in with high fever and vomiting, it may indicate the onset of an exanthem, or occasionally be the primary symptom of encephalitis, or whatever the condition is which causes infantile hemiplegia. When the attack is associated with debility and with rickets the diagnosis is easily made. The carpopedal spasms and pseudo-paralytic rigidity which are often associated with rickets, laryngismus stridulus, and the hydrocephaloid state are usually confined to the hands and arms and are intermittent and usually tonic. The convulsions associated with tumor or those which follow infantile hemiplegia are usually at first Jacksonian in character. After the second year convulsive seizures which come on irregularly without apparent cause and recur while the child is apparently in good health, are likely to prove true epilepsy.

Prognosis.—Convulsions play an important part in infantile mortality. In Morris J. Lewis's table of deaths in children under ten, 8.5 per cent. were ascribed to convulsions. In chronic diarrhœa convulsions are usually of ill omen. Those ushering in fevers are rarely serious, and the same may be said of the fits associated with indigestion and peripheral irritation.

Treatment.—Every source of irritation should be removed. If associated with indigestible food, a prompt emetic should be given, followed by an enema. The teeth should be examined, and if the gum is swollen, hot, and tense, it may be lanced; but never if it looks normal. When seen at first, if the paroxysm is severe, no time should be lost by giving a hot bath, but chloroform should be given at once, and repeated if necessary. A child is so readily put under chloroform and with such a small quantity that this procedure is quite harmless and saves much valuable time. The practice is almost universal of putting the child into a warm bath, and if there is a fever the head may be douched with cold water. The temperature of the bath should not be above 95° or 96°. The very hot bath is not suitable, particularly if the fits are due to indigestion. After the attack an ice-cap may be placed upon the head. If there is much irritability, particularly in rickets and in severe diarrhœa, small doses of opium will be found efficacious. When the convulsions recur after the child comes from under the influence of chloroform it is best to place it rapidly under the influence of opium, which may be given as morphia hypodermically, in doses of from one-twenty-fifth to one-thirtieth of a grain (0.0026 to 0.0022 gm.) for a child of one year. Other remedies recommended are chloral by enema, in 5 grain (0.3 gm.) doses, and nitrite of amyl. After the attack has passed the bromides are useful, of which 5 to 8 grains (0.3 to 0.5 gm.) may be given in a day to a child a year old. Recurring convulsions, particularly if they come on without special cause, should receive the most thorough and careful treatment with bromides. When associated with rickets the treatment should be directed to improving the general condition.

V. EPILEPSY

Definition.—An affection of the nervous system characterized by attacks of unconsciousness, with or without convulsions.

The transient loss of consciousness without convulsive seizures is known

as *petit mal;* the loss of consciousness with general convulsive seizures is known as *grand mal.* Localized convulsions, occurring usually without loss of consciousness, are known as epileptiform, or more frequently as Jacksonian or cortical epilepsy.

Etiology.—Age.—In a large proportion of all cases the disease begins before puberty. Of the 1,450 cases observed by Gowers, in 422 the disease began before the tenth year, and three-fourths of the cases began before the twentieth year. Of 460 cases of epilepsy in children ~~which I have analyzed~~ the age of onset in 427 was as follows: First year, 74; second year, 62; third year, 51; fourth year, 24; fifth year, 17; sixth year, 18; seventh year, 19; eighth year, 23; ninth year, 17; tenth year, 27; eleventh year, 17; twelfth year, 18; thirteenth year, 15; fourteenth year, 21; fifteenth year, 34. Arranged in hemidecades the figures are as follows: From the first to the fifth year, 229; from the fifth to the tenth year, 104; from the tenth to the fifteenth year, 95. These figures illustrate in a striking manner the early onset of the disease in a large proportion of the cases. It is well always to be suspicious of epilepsy beginning in adult life, for in a majority of such cases the convulsions are due to a local lesion.

Sex.—No special influence appears to be discoverable in this relation, certainly not in children. Of 433 cases ~~in my tables~~, 232 were males and 203 were females, showing a slight predominance of the male sex. After puberty unquestionably, if a large number of cases are taken, the males are in excess.

Heredity.—Much stress has been laid upon this by many authors as an important predisposing cause, and the statistics collected give from 9 to over 40 per cent. Gowers gives 35 per cent. for his cases, which have special value apart from other statistics embracing large numbers of epileptics in that they were collected by him in his own practice. In other figures it appears to play a minor rôle. In my list there were only 31 cases in which there was a history of marked neurotic taint, and only 3 in which the mother herself had been epileptic. In the Elwyn cases, as might be expected, the percentage is larger. Of the 126 there was in 32 a family history of nervous derangement of some sort, either paralysis, epilepsy, marked hysteria, or insanity. Spratling found 16 per cent. among 2,523 cases.

While, then, it may be said that direct inheritance is comparatively ~~uncommon~~, yet the children of neurotic families in which neuralgia, insanity, and hysteria prevail are more liable to fall victims to the disease.

Chronic alcoholism in the parents is regarded by many as a potent predisposing factor, ~~in the production of epilepsy~~. Echeverria ~~has~~ analyzed 572 cases ~~bearing upon this point~~ and divided them into three classes, of which 257 cases could be traced directly to alcohol as a cause; 126 cases in which there were associated conditions, such as syphilis and traumatism; 189 cases in which the alcoholism was probably the result of the epilepsy. Figures equally strong are given by Martin, who in 150 insane epileptics found 83 with a marked history of parental intemperance. Spratling found 15 per cent. with marked alcoholic history in the parents.

Syphilis.—This in the parents is probably less a predisposing than an actual cause of epilepsy, which is the direct outcome of local cerebral manifestations. There is no reason for recognizing a special form of syphilitic

epilepsy. On the other hand, convulsive seizures due to acquired syphilitic disease of the brain are very common.

Alcohol.—Severe epileptic convulsions may occur in steady drinkers.

Of exciting causes fright is believed to be important, but is less so, I think, than is usually stated. Trauma is present in a certain number of instances. An important group depends upon a local disease of the brain existing from childhood, as seen in the post-hemiplegic epilepsy. Occasionally cases follow the infectious fevers. Masturbation has been stated to be a special cause, but its influence is probably overrated. A large group of convulsive seizures allied to epilepsy are due to some toxic agent, as in lead poisoning and in uræmia.

Reflex Causes.—Eye strain, dentition, and worms, the irritation of a cicatrix, some local affection, such as adherent prepuce, or a foreign body in the ear or the nose, are given as causes. In some of these cases the fits cease after the removal of the cause, so that there can be no question of the association between the two. In others the attacks persist. Genuine cases of reflex epilepsy are, I believe, rare. A remarkable instance of it occurred at the Philadelphia Infirmary for Diseases of the Nervous System in the case of a man with a testis in the inguinal canal, pressure upon which would cause a typical fit. Removal of the organ was followed by cure.

Cardio-vascular epilepsy is usually a manifestation of advanced arteriosclerosis, and is associated with slow pulse (see Stokes-Adams Disease). There may be palpitation and uneasy sensations about the heart prior to the attack. The passage of a gall-stone or the removal of pleuritic fluid may induce a fit. Indigestion and gastric troubles are extremely common in epilepsy, and in many instances the eating of indigestible articles seems to precipitate an attack. And lastly, epileptic seizures may occur in old people without obvious cause.

Symptoms.—(*a*) Grand Mal.—Preceding the fits there is usually a localized sensation, known as an *aura*, in some part of the body. This may be somatic, in which the feeling comes from some particular region in the periphery, as from the finger or hand, or is a sensation felt in the stomach or about the heart. The peripheral sensations preceding the fit are of great value, particularly those in which the aura always occurs in a definite region, as in one finger or toe. It is the equivalent of the signal symptom in a fit from a brain tumor. The varieties of these sensations are numerous. The epigastric sensations are most common. In these the patient complains of an uneasy sensation in the epigastrium or distress in the intestines, or the sensation may not be unlike that of heartburn and may be associated with palpitation. These groups are sometimes known as pneumogastric auræ or warnings.

Of psychical auræ one of the most common, as described by Hughlings Jackson, is the vague, dreamy state, a sensation of strangeness or sometimes of terror. The auræ may be associated with special senses; of these the most common are the visual, consisting of flashes of light or sensations of color; less commonly, distinct objects are seen. The auditory auræ consist of noises in the ear, odd sounds, musical tones, or occasionally voices. Olfactory and gustatory auræ, unpleasant tastes and odors, are rare.

Occasionally the fit may be preceded not by an aura, but by certain movements; the patient may turn round rapidly or run with great speed for a few

minutes, the so-called epilepsia procursiva. In one of the Elwyn cases the lad stood on his toes and twirled with extraordinary rapidity, so that his features were scarcely recognizable. It is stated that the pulse sometimes stops just before the fit. The studies of Gibson and Good show that no alteration in the pulse occurred up to the point of clonic convulsions, and there was no lowering of the general blood pressure suggesting anæmia of the brain. At the onset of the attack the patient may give a loud scream or yell, the so-called epileptic cry. The patient drops as if shot, making no effort to guard the fall. In consequence of this epileptics frequently injure themselves, cutting the face or head or burning themselves. In the attack, as described by Hippocrates, "the patient loses his speech and chokes, and foam issues from the mouth, the teeth are fixed, the hands are contracted, the eyes distorted, he becomes insensible, and in some cases the bowels are affected. And these symptoms occur sometimes on the left side, sometimes on the right, and sometimes on both." The fit may be described in three stages:

(1) *Tonic Spasm.*—The head is drawn back or to the right, and the jaws are fixed. The hands are clinched and the legs extended. This tonic contraction affects the muscles of the chest, so that respiration is impeded and the initial pallor of the face changes to a dusky or livid hue. The muscles of the two sides are unequally affected, so that the head and neck are rotated or the spine is twisted. The arms are usually flexed at the elbows, the hand at the wrist, and the fingers are tightly clinched in the palm. This stage lasts only a few seconds, and then the clonic stage begins.

(2) *Clonic Stage.*—The muscular contractions become intermittent; at first tremulous or vibratory, they gradually become more rapid and the limbs are jerked and tossed about violently. The muscles of the face are in constant clonic spasm, the eyes roll, the eyelids are opened and closed convulsively. The movements of the muscles of the jaw are very forcible and strong, and it is at this time that the tongue is apt to be caught between the teeth and lacerated. The cyanosis, marked at the end of the tonic stage, gradually lessens. A frothy saliva, which may be blood stained, escapes from the mouth. The fæces and urine may be discharged involuntarily. The duration of this stage is variable. It rarely lasts more than one or two minutes. The contractions become less violent and the patient gradually sinks into the condition of coma.

(3) *Coma.*—The breathing is noisy or even stertorous, the face congested, but no longer intensely cyanotic. The limbs are relaxed and the unconsciousness is profound. After a variable time the patient can be aroused, but if left alone he sleeps for some hours and then awakes, complaining only of slight headache or mental confusion. If the attack has been severe, petechial hæmorrhages may be scattered over the neck and chest. In the case of a young man in good health in a severe convulsion both sub-conjunctival spaces were entirely filled with blood, and free blood oozed from them (Walter James). Hæmoptysis is a rare sequel.

(4) *Status Epilepticus.*—This is the climax of the disease, in which attacks occur in rapid succession, and the patient does not recover consciousness. The pulse, respiration, and temperature rise in the attack. It is a serious condition, and often proves fatal.

After the attack the reflexes are sometimes absent; more frequently they

are increased and the ankle clonus can usually be obtained. The state of the urine is variable, particularly as regards the solids. The quantity is usually increased after the attack, and albumin is not infrequently present.

(5) *Post-epileptic symptoms* are of great importance. The patient may be in a trance-like condition, in which he performs actions of which subsequently he has no recollection. More serious are the attacks of mania, in which the patient is often dangerous and sometimes homicidal. It is held by good authorities that an outbreak of mania may be substituted for the fit. And, lastly, the mental condition of an epileptic patient is often seriously impaired, and profound defects are common.

(6) *Paralysis*, which rarely follows the epileptic fit, is usually hemiplegic and transient. Slight disturbances of speech also may occur; in some instances, forms of sensory aphasia.

The attacks may occur at night, and a person may be epileptic for years without knowing it. As Trousseau truly remarks, when a person tells us that in the night he has incontinence of urine and awakes in the morning with headache and mental confusion, and complains of difficulty in speech owing to the fact that he has bitten his tongue, if also there are purpuric spots on the skin of the face and neck, the probability is very strong indeed that he is subject to nocturnal epilepsy.

(*b*) Petit Mal.—This is epilepsy without the convulsions. The attack consists of transient unconsciousness, which may come on at any time, accompanied or unaccompanied by a feeling of faintness and vertigo. Suddenly, for example, at the dinner table, the subject stops talking and eating, the eyes become fixed, and the face slightly pale. Anything which may have been in the hand is usually dropped. In a moment or two consciousness is regained and the patient resumes conversation as if nothing had happened. In other instances there is slight incoherency or the patient performs some almost automatic action. He may begin to undress himself and on returning to consciousness find that he has partially disrobed. He may rub his beard or face, or may spit about in a careless way. In other attacks the patient may fall without convulsive seizures. A definite aura is rare. Though transient, unconsciousness and giddiness are the most constant manifestations of *petit mal*, there are many other equivalent manifestations, such as sudden jerkings in the limbs, sudden tremor, or a sudden visual sensation. Gowers mentions no less than seventeen different manifestations of *petit mal*. Occasionally there are cases in which the patient has a sensation of losing his breath and may even get red in the face.

After the attack the patient may be dazed for a few seconds and perform certain automatic actions, which may seem to be volitional. As mentioned, undressing is a common action, but all sorts of odd actions may be performed, some of which are awkward or even serious. One of my patients after an attack was in the habit of tearing anything he could lay hands on, particularly books. Violent actions have been committed and assaults made, frequently giving rise to questions which come before the courts. This condition has been termed masked epilepsy, or *epilepsia larvata*.

In a majority of the cases of *petit mal* convulsions finally occur, at first slight, but ultimately the *grand mal* becomes well developed, and the attacks may then alternate.

(*c*) Jacksonian Epilepsy.—This is also known as cortical, symptomatic, or partial epilepsy. It is distinguished from the ordinary epilepsy by the important fact that consciousness is retained or is lost late. The attacks are usually the result of irritative lesions in the motor zone, though there are probably also sensory equivalents of this motor form. In a typical attack the spasm begins in a limited muscle group of the face, arm, or leg. The zygomatic muscles, for instance, or the thumb may twitch, or the toes may first be moved. Prior to the twitching the patient may feel a sensation of numbness or tingling in the part affected. The spasm extends and may involve the muscles of one limb only or of the face. The patient is conscious throughout and watches, often with interest, the march of the spasm.

The onset may be slow, and, as in a case which I have reported, there may be time for the patient to place a pillow on the floor, so as to be as comfortable as possible during the attack. The spasms may be localized for years, but there is a great risk that the partial epilepsy may become general. The condition is due, as a rule, to an irritative lesion in the motor zone. Thus of 107 cases analyzed by Roland, there were 48 of tumor, 21 instances of inflammatory softening, 14 instances of acute and chronic meningitis, and 8 cases of trauma. The remaining instances were due to hæmorrhage or abscess, or were associated with sclerosis cerebri. Two other conditions may be mentioned, which may cause typical Jacksonian epilepsy—namely, uræmia and progressive paralysis of the insane. A considerable number of the cases of Jacksonian epilepsy are found in children following hemiplegia, the so-called post-hemiplegic epilepsy. The convulsions usually begin on the affected side, either in the arm or leg, and the fit may be unilateral and without loss of consciousness. Ultimately they become more severe and general.

Diagnosis.—In *major epilepsy* the suddenness of the attack, the abrupt loss of consciousness, the order of the tonic and clonic spasm, and the relaxation of the sphincters at the height of the attack are distinctive features. The convulsive seizures due to uræmia are epileptic in character and usually readily recognized by the existence of greatly increased tension and the condition of the urine. Practically in young adults hysteria causes the greatest difficulty, and may closely simulate true epilepsy. The table on page 1085, from Gowers' work, draws clearly the chief differences between them.

Recurring epileptic seizures in a person over thirty who has not had previous attacks is always suggestive of organic disease. According to H. C. Wood, whose opinion is supported by that of Fournier, in 9 cases out of 10 the condition is due to syphilis.

Petit mal must be distinguished from attacks of syncope, and the vertigo of Ménière's disease, of a cardiac lesion, and of indigestion. In these cases there is no actual loss of consciousness, which forms a characteristic though not an invariable feature of *petit mal*.

Jacksonian epilepsy has features so distinctive and peculiar that it is at once recognized. It is, however, by no means easy always to determine upon what the spasm depends. Irritation in the motor centres may be due to a great variety of causes, among which tumors and localized meningo-encephalitis are the most frequent; but it must not be forgotten that in uræmia localized epilepsy may occur. The most typical Jacksonian spasms also are not infrequent in general paresis of the insane.

	Epileptic	Hysteroid
Apparent cause	none.	emotion.
Warning	any, but especially unilateral or epigastric auræ.	palpitation, malaise, choking, bilateral foot aura.
Onset	always sudden.	often gradual.
Scream	at onset.	during course.
Convulsion	rigidity followed by "jerking," rarely rigidity alone.	rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting	tongue.	lips, hands, or other people and things.
Micturition	frequent.	never.
Defecation	occasional.	never.
Talking	never.	frequent.
Duration	a few minutes.	more than ten minutes, often much longer.
Restraint necessary	to prevent accident.	to control violence.
Termination	spontaneous.	spontaneous or induced (water, etc.).

Prognosis.—This may be given to-day in the words of Hippocrates: "The prognosis in epilepsy is unfavorable when the disease is congenital, and when it endures to manhood, and when it occurs in a grown person without any previous cause. . . . The cure may be attempted in young persons, but not in old." W. A. Turner concludes that of cases beginning under ten years few are arrested, whereas of those beginning at puberty the opposite is true. Cases beginning between the twentieth and thirty-fifth years give few arrests. After thirty-five the outlook is good.

Death during the fit rarely occurs, but it may happen if the patient falls into the water or if the fit comes on while he is eating. Occasionally the fits seem to stop spontaneously. This is particularly the case in the epilepsy in children which has followed the convulsions of teething or of the fevers. Frequency of the attacks and marked mental disturbance are unfavorable indications. Hereditary predisposition is apparently of no moment in the prognosis. The outlook is better in males than in females. The post-hemiplegic epilepsy is rarely arrested. Of the cases coming on in adults, those due to syphilis and to local affections of the brain allow a more favorable prognosis.

Treatment.—General.—In the case of children the parents should be made to understand from the outset that epilepsy in the great majority of cases is an incurable affection, so that the disease may interfere as little as possible with the education of the child. The subjects need firm but kind treatment. Indulgence and yielding to caprices and whims are followed by weakening of the moral control, which is so necessary in these cases. The disease does not incapacitate a person for all occupation. It is much better for epileptics to have some definite pursuit. There are many instances in which they have been persons of extraordinary mental and bodily vigor, as, for example, Julius Cæsar and Napoleon. One of the most distressing features in epilepsy is the gradual mental impairment which follows in a certain number of cases. If such patients become extremely irritable or show signs of violence they should be placed under supervision in an asylum. Marriage should be forbidden to epileptics. During the attack a cork or bit of rubber

should be placed between the teeth and the clothes should be loosened. The patient should be in the recumbent posture. As the attack usually passes off with rapidity, no special treatment is necessary, but in cases in which the convulsion is prolonged a few whiffs of chloroform or nitrite of amyl or a hypodermic of a quarter of a grain of morphia may be given.

Dietetic.—The old authors laid great stress upon regimen in epilepsy. The important point is to give the patient a light diet at fixed hours, and on no account to permit overloading of the stomach. Meat should not be given more than once a day. There are cases in which animal food seems injurious. A strict vegetable diet has been warmly recommended. The patient should not go to sleep until the completion of gastric digestion.

Medicinal.—The bromides are the only remedies which have a special influence upon the disease. Either the sodium or potassium salt may be given. Sodium bromide is probably less irritating and is better borne for a long period. It may be given in milk, in which it is scarcely tasted. In all instances the dilution should be considerable. In adults it is well taken in soda water or in some mineral water. The dose for an adult should be from half a drachm to a drachm and a half (2 to 6 gm.) daily. As Seguin recommends, it is often best to give but a single dose daily, about four to six hours before the attacks are most likely to occur. For instance, in the case of nocturnal epilepsy a drachm should be given an hour or two after the evening meal. If the attack occurs early in the morning, the patient should take a full dose when he awakes. When given three times a day it is less disturbing after meals. Each case should be carefully studied to determine how much bromide should be used. The individual susceptibility varies and some patients require more than others. Fortunately, children take the drug well and stand proportionately larger doses than adults. Saturation is indicated by certain unpleasant effects, particularly drowsiness, mental torpor, and gastric and cardiac distress. Loss of palate reflex is one of the earliest indications that the system is under the influence of the bromides, and is a condition which should be attained. A very unpleasant feature is the development of acne, which, however, is no indication of bromism. Seguin states that the tendency to this is much diminished by giving the drug largely diluted in alkaline waters and administering from time to time full doses of arsenic. To be effectual the treatment should be continued for a prolonged period and the cases should be incessantly watched in order to prevent bromism. The medicine should be continued for at least two years after the cessation of the fits; indeed, Seguin recommends that the reduction of the bromides should not be begun until the patient has been three years without any manifestations. Written directions should be given to the mother or to the friends of the patient, and he should not himself be held responsible for the administration of the medicine. A book should be provided in which the daily number of attacks and the amount of medicine taken should be noted. The addition of belladonna to the bromide is warmly recommended by Black, of Glasgow. In very obstinate cases Flechsig uses opium, 5 or 6 grains (0.35 gm.), in three doses daily; then at the end of six weeks opium is stopped and the bromides in large amounts, 75 to 100 grains (4 to 6 gm.) daily, are used for two months.

Among other remedies which have been recommended as controlling epi-

lepsy are chloral, cannabis indica, zinc, nitroglycerin, and borax. Nitroglycerin is sometimes advantageous in *petit mal*, but is not of much service in the major form. To be beneficial it must be given in full doses, from 2 to 5 drops of the 1 per cent. solution, and increased until the physiological effects are produced. Calcium lactate in 20 grain (1.3 gm.) doses daily has been highly recommended. Counter-irritation is rarely advisable. When the aura is very definite and constant in its onset, as from the hand or from the toe, a blister about the part or a ligature tightly applied may stop the oncoming fit. In children, care should be taken that there is no source of peripheral irritation. In boys, adherent prepuce may occasionally be the cause. The irritation of teething, the presence of worms, and foreign bodies in the ears or nose have been associated with epileptic seizures.

The subjects of a chronic and, in most cases, a hopelessly incurable disease, epileptic patients form no small portion of the unfortunate victims of charlatans and quacks, who prescribe to-day, as in the time of the father of medicine, "purifications and spells and other illiberal practices of like kind."

Surgical.—In Jacksonian epilepsy the propriety of surgical interference is universally granted. It is questionable, however, whether in the epilepsy following hemiplegia, considering the anatomical condition, it is likely to be of any benefit. In idiopathic epilepsy, when the fit starts in a certain region —the thumb, for instance—and the signal symptom is invariable, the centre controlling this part may be removed. Operation in the traumatic epilepsy, in which the fit follows fracture, is much more hopeful.

The operation, *per se*, appears in some cases to have a curative effect. Thus of 50 cases of trephining for epilepsy in which nothing abnormal was found to account for the symptoms, 25 were reported as cured and 18 as improved. The operations have not been always on the skull, and White has collected an interesting series in which various surgical procedures have been resorted to, often with curative effect, such as ligation of the carotid artery, castration, tracheotomy, excision of the superior cervical ganglia, incision of the scalp, circumcision, etc.

VI. MIGRAINE

(*Hemicrania; Sick Headache*)

Definition.—A paroxysmal affection characterized by severe headache, usually unilateral, and often associated with disorders of vision.

Etiology.—Heredity plays an important rôle in 90 per cent. of cases according to Möbius. Women and members of neurotic families are most frequently attacked. Many distinguished men have been its victims, and the astronomer Airy gave a classical account of his case. The nature of the disease is unknown, and many views have been entertained:

(*a*) That it is a toxæmia from disorder of the intestinal digestion, from disturbed uric acid output, or from some self-manufactured poison.

(*b*) That it is a vasomotor affection with spasm of the arteries, in favor of which are the facts that in the attack the temporal arteries on the affected side may be felt to be small, the retinal arteries may sometimes be seen in spasm, and sclerosis of the arteries on the same side is found in a certain

number of cases of hemicrania. A still more striking confirmation is the temporary paralysis which may be associated with an attack of monoplegic or hemiplegic character. Mitchell Clarke has reported a history of recurring motor paralysis in eleven members in three generations of the same family. The characteristic visual phenomena preceded the unilateral headache, especially the hemiopia. In most of the attacks the hemiplegia was on the right side. It lasted from a few hours to a day and disappeared completely, leaving no damage. It is difficult to explain such cases except on the view of a transient spasm of the arteries.

(*c*) Others regard the affection as of reflex origin arising from a refractive error in the eyes, or from troubles in the nose or sexual organs.

(*d*) The disease has been attributed to transient plugging of the foramen of Monro with increased pressure in the ventricles (Spitzner).

The majority of cases begin early in life, and Sinclair refers to a case in a child of two years. Many circumstances bring on the attack: a powerful emotion of any sort, mental or bodily fatigue, digestive disturbances, or the eating of some particular article of food. The paroxysmal character is one of the most striking features of the attacks which may occur on the same day every week, every fortnight, or every month. Headaches of the migraine type may occur for years in connection with chronic nephritis, and it is well to remember that attacks may occur in connection with tumors and other lesions of the base of the brain.

Symptoms.—Premonitory signs are present in many cases, and the patient can tell when an attack is coming on. Remarkable prodromata have been described, particularly in connection with vision. Apparitions may appear—visions of animals, such as mice, dogs, etc. Transient hemianopia or scotoma may be present. In other instances there is spasmodic action of the pupil on the affected side, which dilates and contracts alternately, the condition known as *hippus*. Frequently the disturbance of vision is only a blurring, or there are balls of light, or zigzag lines, or the so-called fortification spectra (teichopsia), which may be illuminated with gorgeous colors. Disturbances of the other senses are rare. Numbness of the tongue and face and occasionally of the hand may occur with tingling. More rarely there are cramps or spasms in the muscles of the affected side. Transient aphasia has also been noted, which may be intermittent. Some patients show marked psychical disturbance, either excitement or, more commonly, mental confusion or great depression. Dizziness occurs in some cases. The headache follows a short time after the prodromal symptoms have appeared. It is cumulative and expansile in character, beginning as a localized small spot, which is generally constant either on the temple or forehead or in the eyeball. It is usually described as of a penetrating, sharp, boring character. The pain gradually spreads and involves the entire side of the head, sometimes the neck, and may pass into the arm. In some cases both sides are affected. Nausea and vomiting are common symptoms. If the attack comes on when the stomach is full vomiting usually gives relief. Vasomotor symptoms may be present. The face, for instance, may be pale, and there may be a marked difference between the two sides. Subsequently the face and ear on the affected side may become a burning red from the vaso-dilator influences. The pulse may be slow. The temporal artery on the affected side may be firm and hard, and in a condition of

arterio-sclerosis—a fact which has been confirmed anatomically by Thoma. Few affections are more prostrating than migraine, and during the paroxysm the patient may scarcely be able to raise the head from the pillow. The slightest noise or light aggravates the condition.

The duration of the entire attack is variable. The severer forms usually incapacitate the patient for at least three days. In other instances the entire attack is over in a day. The disease recurs for years, and in cases with a marked hereditary tendency may persist throughout life. In women the attacks often cease after the climacteric, and in men after the age of fifty. Two of the greatest sufferers I have known, who had recurring attacks every few weeks from early boyhood, now have complete freedom.

Treatment.—The patient is usually aware of the causes which precipitate an attack. Avoidance of excitement, regularity in the meals, and moderation in diet are important rules. I have known cases greatly benefited by a strict vegetable diet. The treatment should be directed toward the removal of the conditions upon which the attacks depend. In children much may be done by watchfulness and care on the part of the mother in regulating the bowels and watching the diet of the child. Errors of refraction should be adjusted. On no account should such children be allowed to compete in school for prizes. A prolonged course of bromides sometimes proves successful. If anæmia is present, iron and arsenic should be given. When the arterial tension is increased a course of nitroglycerin may be tried. Not too much, however, should be expected of the preventive treatment of migraine. In a very large proportion of the cases the headaches recur in spite of all we (including the refractionists) can do. Harter advised, so soon as the patient has any intimation of the attack, to wash out the stomach with water at 105°, and to give a brisk saline cathartic. Irrigation of the colon with hot saline solution is sometimes of value if done at the onset. Alkaline water should be taken freely by mouth. During the paroxysm the patient should be kept in bed and absolutely quiet. If the patient feels faint and nauseated a small cup of hot, strong coffee or 20 drops of chloroform give relief. Cannabis indica is probably the most satisfactory remedy. Seguin recommends a prolonged course of the drug. Antipyrin, antifebrin, and phenacetin have been much used of late. When given early, at the very outset of the paroxysm, they are sometimes effective. Small, repeated doses are more satisfactory. Of other remedies, caffeine, in 5-grain doses of the citrate, nux vomica, and ergot have been recommended. Electricity does not appear to be of much service.

Ophthalmoplegic Migraine.—This term was applied by Charcot to a special form in which there is weakness or paralysis of one or more eye muscles, with or after a migraine attack. The oculo-motor nerve is usually involved. Ptosis, loss of certain movements, and double vision are the common features, which may persist for some days. Local causes, especially syphilis, should be excluded before the diagnosis is established. The treatment is the same as for migraine.

VII. NEURALGIA

Definition.—A painful affection of the nerves, due either to functional disturbance of their central or peripheral extremities or to neuritis in their course.

Etiology.—Members of neuropathic families are most subject to the disease. It affects women more than men. Children are rarely attacked. Of all causes debility is the most frequent. It is often the first indication of an enfeebled nervous system. The various forms of anæmia are frequently associated with neuralgia. It may be a prominent feature at the onset of certain acute diseases, particularly typhoid fever. Malaria has been thought to be a potent cause, but it has not been shown that neuralgia is more frequent in malarial districts, and the error has probably arisen from regarding periodicity as a special manifestation of paludism. It occasionally occurs in malarial cachexia. Exposure to cold is a cause in very susceptible persons. Reflex irritation, particularly from carious teeth, and disease of the antrum and frontal sinuses are common causes of neuralgia of the fifth nerve. The disease occurs sometimes in gout, lead poisoning, and diabetes. Persistent neuralgia may be a feature of latent Bright's disease.

Symptoms.—Before the onset of the pain there may be uneasy sensations, sometimes tingling in the part which will be affected. The pain is localized to a certain group or division of nerves, usually affecting one side. The pain is not constant, but paroxysmal, and is described as stabbing, burning, or darting in character. The skin may be exquisitely tender in the affected region, particularly over certain points along the course of the nerve, the so-called tender points. Movements, as a rule, are painful. Trophic and vaso-motor changes may accompany the paroxysm; the skin may be cool, and subsequently hot and burning; occasionally local œdema or erythema occurs. More remarkable still are the changes in the hair, which may become blanched (canities), or even fall out. Fortunately, such alterations are rare. Twitchings of the muscles, or even spasms, may be present during the paroxysm. After lasting a variable time—from a few minutes to many hours—the attack subsides. Recurrence may be at definite intervals—every day at the same hour, or at intervals of two, three, or even seven days. Occasionally the paroxysms develop only at the catamenia. This periodicity is quite as marked in non-malarial as in malarial regions.

CLINICAL VARIETIES, DEPENDING ON THE NERVE ROOTS AFFECTED

Trigeminal Neuralgia; Tic Douloureux.—A distinction must be drawn between the minor and major neuralgias of the fifth cranial nerve. The former may merely be symptomatic of the involvement of one or another of its peripheral branches in some disease process—the pressure of a tumor, carious teeth, or a neuritis due to the proximity of suppurative processes in the bony sinuses, etc. There may be referred neuralgic pains in this area from morbid processes within the cranium, or from visceral disease elsewhere. A painful neuralgia may follow an attack of zoster in any division of the fifth nerve.

The typical *tic douloureux, epileptiform neuralgia,* or *"neuralgia quinti major,"* as it has been called, is probably a primary affection of the Gasserian ganglion. The disease starts in middle life, without obvious cause, as a simple neuralgia in one of the trigeminal branches, and from a particular spot the pain radiates through the course of one of the nerves, most often the upper branch. The pain is of sudden onset, violent and paroxysmal in character. There are periods of remission, which at first may extend over several months,

Patrick. J.A.M.A. May 16th '14.

Sexes equal about. age - nearly 60% between 40 & 60.

no def. viol fever but not so hard mig[illegible]

Side. right in nearly 2/3.

Branch. first - 18.5. II. 43.6. III. 37.5. -

Regain in I. 8.5%
II 45.
III. 25

Recurrence 7.5%

pain limited to supra-orbital region is probably not neuralgia.

Pain. short - paroxysm rarely over 2 min. may be a succession

∴ any pain about face wh. is not already ache - not trifacial neuralgia

(the exceptions)

Severe usually unconcealing.

Starts by relatively slight peripheral irritation

doloroquendi zones - an area in which slight irritation

after small ones at first. starts pain.

"trigger" zone.

pain-genetic & pain zones may not correspond.

may be of distribution of different divisions

(mem. in treatment - better to attack "trigger" zone first.

active no. of divisions I 47%
two 43%
three 10%

So if touches face to shave side of pain - not has been rubbing with towel or massage - not. eating, chewing, washing will set pain - not.

pain may radiate into cervical nerves & down arm

Tender tonsils of Vallecula — Do not exist. (They can be seen in other adults
if depressed)

Dura chronic — persistent but also remittent. — may be for years
tender to pain or worse.

Neuromata traumatic — not rare. may be carcinomata or sarcomata

Tic douloureux — a misnomer. Drop — immobility muscular
spasm, twitching rare. — many causes spinal nerves

Lumbalgia: syphilis. very rare — ? acute ri[illegible] — e.g. hyperesthesia
cutaneous

Testis tender. not frequent — malaria — not.

Appendix + special senses — not rare. no selection [illegible]

and in which the paroxysms do not occur, but these intervals of release shorten after each successive attack. The attacks themselves are of ever increasing severity and longer duration. The pain finally invades the territory of adjoining nerves and ultimately, after years, may extend over the entire trigeminal distribution. Though by sympathy there may be pain outside of the fifth nerve area, particularly in the occipital region, in true tic douloureux the pain remains limited to the distribution of one trigeminal nerve, and probably never becomes bilateral. In advanced cases the paroxysms follow one another rapidly and without assignable cause, and in the intervals the patient may never be quite free from pain. They are inaugurated by almost any form of external stimulus, by a draught of air, by movement of the facial muscles or of the tongue in speaking, by touching the skin, particularly over those points from which the pain seems to take its origin, by the act of swallowing, especially when the pain involves the mucous membrane field of distribution of the nerve. It is not a self-limited disease. In some instances the neuralgia reaches such a frightful intensity that it renders the patient's life insupportable. In former years suicide was not an uncommon consequence.

Cervico-occipital neuralgia involves the posterior branches of the first four cervical nerves, particularly the inferior occipital, at the emergence of which there is a painful point about half-way between the mastoid process and the first cervical vertebra. It may be caused by cold, and these nerves are often affected in cervical caries. Surgical measures may be required if the pain is severe. Krause has devised an operation for division and evulsion of the affected nerves.

Cervico-brachial neuralgia involves the sensory nerves of the brachial plexus, particularly in the cubital division. When the circumflex nerve is involved the pain is in the deltoid. The pain is most commonly about the shoulder and down the course of the ulnar nerve. There is usually a marked tender point upon this nerve at the elbow. This form rarely follows cold, but more frequently results from rheumatic affections of the joints, and trauma.

Neuralgia of the phrenic nerve is rare. It is sometimes found in pleurisy and in pericarditis. The pain is chiefly at the lower part of the thorax on a line with the insertion of the diaphragm, and here may be painful points on deep pressure. Full inspiration is painful, and there is great sensitiveness on coughing or in the performance of any movement by which the diaphragm is suddenly depressed.

Intercostal Neuralgia.—Next to the *tic douloureux* this is the most important form. It is most frequent in women and very common in hysteria. Post-zoster neuralgias are common in this situation. The possibility of spinal disease, of tumor, caries, or aneurism must always be borne in mind.

Lumbar Neuralgia.—The affected nerves are the posterior fibres of the lumbar plexus, particularly the ilio-scrotal branch. The pain is in the region of the iliac crest, along the inguinal canal, in the spermatic cord, and in the scrotum or labium majus. The affection known as irritable testis, probably a neuralgia of this nerve, may be very severe and accompanied by syncopal sensations.

Coccydynia.—This is regarded as a neuralgia of the coccygeal plexus.

It is most common in women, and is aggravated by the sitting posture. It is very intractable, and may necessitate the removal of the coccyx, an operation, however, which is not always successful. Neuralgias of the nerves of the leg have already been considered.

Neuralgias of the Nerves of the Feet.—Many of these cases accompany varying degrees of flat-foot. The condition is brought about by weakness or fatigue of the muscles supporting the arches of the foot, which consequently settle until the strain of the superimposed body-weight falls upon the ligamentous and aponeurotic attachments between the metatarsal and tarsal bones. Rest, massage, exercises, and orthopædic measures are indicated.

Painful Heel.—Both in women and men there may be about the heel severe pains which interfere seriously with walking—the pododynia of S. D. Gross. There may be little or no swelling, no discoloration, and no affection of the joints. Some cases follow a gonococcus infection and are due to a bony spur.

Plantar Neuralgia.—This is often associated with a definite neuritis, such as follows typhoid fever, and has been seen in an aggravated form in caisson disease (Hughes). The pain may be limited to the tips of the toes or to the ball of the great toe. Numbness, tingling, and hyperæsthesia or sweating may occur with it. In typhoid fever it is not uncommon for patients to complain of great sensitiveness in the toes.

Metatarsalgia.—Thomas G. Morton's "painful affection of the fourth metatarso-phalangeal articulation" is a peculiar and very trying disorder, seen most frequently in women, and usually in one foot. Morton regards it as due to a pinching of the metatarsal nerve. The condition usually requires operation. The red, painful neuralgia—erythromelalgia—is described under the vaso-motor and trophic disturbances.

Visceral Neuralgias.—The more important of these have already been referred to in connection with the cardiac and the gastric neuroses. They are most frequent in women, and are constant accompaniments of neurasthenia and hysteria. The pains are most common in the pelvic region, particularly about the ovaries. Nephralgia is of great interest, for, as has already been mentioned, the symptoms may closely simulate those of stone.

TREATMENT OF NEURALGIA

In general, causes of reflex irritation should be carefully removed. The neuralgia, as a rule, recurs unless the general health improves; so that tonic and hygienic measures of all sorts should be employed. Often a change of air or surroundings will relieve a severe neuralgia. I have known obstinate cases to be cured by a prolonged residence in the mountains, with an out-of-door life and plenty of exercise. A strict vegetable diet will sometimes relieve the neuralgia or headache of a gouty person. Of general remedies, iron is often a specific in the cases associated with chlorosis and anæmia. Arsenic, too, is very beneficial in these forms, and should be given in ascending doses. The value of quinine has been much overrated. It probably has no more influence than any other bitter tonic, except in the rare instances in which the neuralgia is definitely associated with malarial poisoning. Strychnine, cod-liver oil, and phosphorus are also advantageous. Of remedies for the pain, antipyrin, anti-

febrin, and phenacetin should first be tried, for they are sometimes of service. Morphia should be given with great caution, and only after other remedies have been tried in vain. On no consideration should the patient be allowed to use the hypodermic syringe. Gelsemium is highly recommended. Of nerve stimulants, valerian and ether, which often act well together, may be given. Alcohol is a valuable though dangerous remedy, and should not be ordered for women. In the minor form of trigeminal neuralgia nitroglycerin in large doses may be tried. Dana has seen good results follow rest with large doses of strychnia given hypodermically. Aconitin in doses of one two-hundredth of a grain (0.00032 gm.) may be tried.

Of local applications, the thermo-cautery is invaluable, particularly in zona and the more chronic forms of neuralgia. Acupuncture may be used. Chloroform liniment, camphor and chloral, menthol, the oleates of morphia, atropia, and belladonna used with lanolin may be tried. Freezing over the tender point with ether spray is sometimes successful. The continuous current may be used. The sponges should be warm, and the positive pole should be placed near the seat of the pain. The strength of the current should be such as to cause a slight tingling or burning, but not pain.

For the trigeminal neuralgia cutting of the nerves and removal of the Gasserian ganglion are practised. Alcohol injections into the nerve trunks have been extensively used, and in some hands with excellent results. The nerve fibres are destroyed with total loss of the function of the nerve until regeneration occurs. Wilfred Harris has destroyed the Gasserian ganglion by injecting alcohol into it through the foramen ovale. One of his patients treated in this way thirteen months before has remained perfectly well, and the fifth nerve area is completely anæsthetic. Removal of the ganglion is very satisfactory in skilled hands. Cushing has operated on 130 cases with two deaths, both cases early in the series.

VIII. PROFESSIONAL SPASMS; OCCUPATION NEUROSES

The continuous and excessive use of the muscles in performing a certain movement may be followed by an irregular, involuntary spasm or cramp, which may completely check the performance of the action. The condition is found most frequently in writers, hence the term writer's cramp or scrivener's palsy; but it is also common in piano and violin players and in telegraph operators. The spasms occur in many other persons, such as milkmaids, weavers, and cigarette-rollers.

The most common form is writer's cramp, which is much more frequent in men than in women. Of 75 cases of impaired writing power reported by Poore, all of the instances of undoubted writer's cramp were in men. Morris J. Lewis states that in the United States, in the telegrapher's cramp, women, who are employed a great deal in telegraphy, are much less frequently affected (only 4 out of 43 cases). An investigation by H. Theodore Thompson and J. Sinclair into telegraphist cramp in England shows that the disease is rare, only 13 cases among between 7,000 and 8,000 employees. Persons of a nervous temperament are more liable to the disease. Occasionally it follows slight injury.

Gowers states that in a majority of the cases a faulty method of writing has been employed, using either the little finger or the wrist as the fixed point. Persons who write with the middle of the forearm or the elbow as the fixed point are rarely affected.

No anatomical changes have been found. The most reasonable explanation of the disease is that it results from a deranged action of the nerve centres presiding over the muscular movements involved in the act of writing, a condition which has been termed irritable weakness. "The education of centres which may be widely separated from each other for the performance of any delicate movement is mainly accomplished by lessening the lines of resistance between them, so that the movement, which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this lessened resistance be carried too far, there is an increased and irregular discharge of nerve energy which gives rise to spasm and disordered movement. According to this view, the muscular weakness is explained by an impairment of nutrition accompanying that of function, and the diminished faradic excitability by the nutritional disturbance descending the motor nerves" (Gay).

Symptoms.—These may be described under five heads (Lewis).

(*a*) Cramp or Spasm.—This is often an early symptom and most commonly affects the forefinger and thumb; or there may be a combined movement of flexion and adduction of the thumb, so that the pen may be twisted from the grasp and thrown to some distance. Weir Mitchell has described a lock-spasm, in which the fingers become so firmly contracted upon the pen that it can not be removed.

(*b*) Paresis and Paralysis.—This may occur with the spasm or alone. The patient feels a sense of weakness and debility in the muscles of the hand and arm and holds the pen feebly. Yet in these circumstances the grasp of the hand may be strong and there may be no paralysis for ordinary acts.

(*c*) Tremor.—This is most commonly seen in the forefinger and may be a premonitory symptom of atrophy. It is not an important symptom, and is rarely sufficient to produce disability.

(*d*) Pain.—Abnormal sensations, particularly a tired feeling in the muscles, are very constantly present. Actual pain is rare, but there may be irregular shooting pains in the arm. Numbness or soreness may exist. If, as sometimes happens, a subacute neuritis develops, there may be pain over the nerves and numbness or tingling in the fingers.

(*e*) Vaso-motor Disturbances.—These may occur in severe cases. There may be hyperæsthesia. Occasionally the skin becomes glossy, or there is a condition of local asphyxia resembling chilblains. In attempting to write, the hand and arm may become flushed and hot and the veins increased in size. Early in the disease the electrical reactions are normal, but in advanced cases there may be diminution of faradic and sometimes increase in the galvanic irritability.

Diagnosis.—A well marked case of writer's cramp or palsy could scarcely be mistaken for any other affection. Care must be taken to exclude the existence of any cerebro-spinal disease, such as progressive muscular atrophy or hemiplegia. The physician is sometimes consulted by nervous persons who

fancy they are becoming subject to the disease and complain of stiffness or weakness without displaying any characteristic features.

Prognosis.—The course of the disease is usually chronic. If taken in time and if the hand is allowed perfect rest, the condition may improve rapidly, but too often there is a strong tendency to recurrence. The patient may learn to write with the left hand, but this also may after a time be attacked.

Treatment.—Various prophylactic measures have been advised. ~~As mentioned,~~ it is important that a proper method of writing be adopted. Gowers suggests that if all persons wrote from the shoulder writer's cramp would practically not occur. Various devices have been invented for relieving the fatigue, but none of them are very satisfactory. The use of the type-writer has diminished very much the frequency of scrivener's palsy. Rest is essential. No measures are of value without this. Massage and manipulation, when combined with systematic gymnastics, give the best results. The patient should systematically practise the opposite movements to those concerned in the cramp. This muscle training often gives good results. Poore recommends the galvanic current applied to the muscles, which are at the same time rhythmically exercised. In very obstinate cases the condition remains incurable. ~~I saw a few years ago a~~ A distinguished gynæcologist who had ~~had~~ writer's cramp twenty years ~~before~~, and who had tried all sorts of treatment, including Wolff's method, without any avail. ~~He still has it in aggravated form, but he can~~ do all the finer manipulations of operative work without any difficulty.

The nutrition of the patients is apt to be much impaired, and cod-liver oil, strychnia, and other tonics will be found advantageous. Local applications are of little benefit. Tenotomy and nerve stretching have been abandoned.

IX. HYSTERIA

Definition.—A disorder, chiefly of young women, in which emotional states control the body, leading to perversion of mental, sensory, motor and secretory functions.

Etiology.—Many and diverse views have prevailed since the Greeks ascribed the disease to the vague desires and wanderings of the womb, after which they named it.

Charcot and his followers regarded hysteria as a psychosis, in which morbid states are induced by ideas. The capability of responding to suggestion is the test of its existence. It is a disturbance in the sphere of personality, in which the emotions have an exaggerated influence on the sensory, motor and secretory functions.

Babinski, in a modification of this view, holds that hysteria is a mental condition with certain primary phenomena and certain secondary accidental symptoms. The essence of the primary features is that they may be produced by suggestion, and they may be made to disappear by persuasion (pithiatism). The primary symptoms include such features as hemi-anæsthesia, paralysis, contractures, etc.; secondary features, as for example muscular atrophy, are directly dependent upon the primary and cannot themselves be induced by suggestion.

In the Breuer-Freud theory, now the vogue, we return to the days of Aretæus, who originated (?) the views of sexual hysteria and believed the womb, "like an animal within an animal" and altogether erratic, caused all sorts of trouble in its wanderings. Freud's view is thus analyzed by Jelliffe in his article in my "System of Medicine," Vol. VII., page 817: "There develop usually on a constitutional basis, in the period before puberty, definite sexual activities which are mostly of a perverse nature. These activities do not, as a rule, lead to a definite neurosis up to the time of puberty, which in the psychic sphere appears much earlier than in the body, but sexual phantasy maintains a perverse constellated direction by reason of the infantile sexual activities. On constitutional (affect) grounds the increased fantasy of the hysteric leads to the formation of complexes which are not taken up by the personality and by reason of shame or disgust remain buried. There, therefore, results a conflict between the characteristic normal libido and the sexual repressions of these buried infantile perversions. These conflicts give rise to the hysterical symptoms. It is in his contributions to the sexual theory that Freud develops his later thoughts of the sexual origin of the hysterical reaction. By sexual it is important to remember that Freud is not speaking of sensual.

"The significance of Freud's theory is the tracing of every case to sexual traumata during early childhood. Sexual experiences differ, however, from ordinary experiences—the latter have a tendency to fade out, while the idea of the former grows with increasing sexual maturity. There results a disproportionate capacity for increased reaction which takes place in the subconscious. This is the cause of the mischief.

"There must be, however, a connecting link between the infantile sexual traumata and the later manifestations. This connection Freud finds in the so-called 'hysterical fancies.' These are the day-dreams of erotic coloring, wish-gratifications, originating in privation and longing. These fancies hark back to the original traumatic moment, and, either originating in the subconscious or shortly becoming conscious, are transformed into hysterical symptoms. They constitute a defence of the ego against the revival, as reminiscences, of the repressed traumatic experiences of childhood" (White).

The affection is most common in women, and usually appears first about the time of puberty, but the manifestations may continue until the menopause, or even until old age. Men, however, are by no means exempt, and hysteria in the male is not rare. It occurs in all races, but is much more prevalent, particularly in its severer forms, in members of the Latin race. In England and the United States the milder grades are common, but the graver forms are rare in comparison with the frequency with which they are seen in France.

Children under twelve years of age are not very often affected, but the disease may be well marked as early as the fifth or sixth year. One of the saddest chapters in the history of human deception, that of the Salem witches, might be headed *hysteria in children*, since the tragedy resulted directly from the hysterical pranks of girls under twelve years of age.

Of predisposing causes, two are important—heredity and education. The former acts by endowing the child with a mobile, abnormally sensitive nervous organization. We see cases most frequently in families with marked neuropathic tendencies, the members of which have suffered from neuroses of vari-

ous sorts. Education at home too often fails to inculcate habits of self control. A child grows to girlhood with an entirely erroneous idea of her relations to others, and accustomed to have every whim gratified and abundant sympathy lavished on every woe, however trifling; she reaches womanhood with a moral organization unfitted to withstand the cares and worries of every-day life. At school, between the ages of twelve and fifteen, the most important period in her life, when the vital energies are absorbed in the rapid development of the body, she is often cramming for examinations and cooped in close school rooms for six or eight hours daily. The result too frequently is an active, bright mind in an enfeebled body, ill adapted to subserve the functions for which it was framed, easily disordered, and prone to react abnormally to the ordinary stimuli of life. Among the more direct influences are emotions of various kinds, fright occasionally, more frequently love affairs, grief, and domestic worries. Physical causes less often bring on hysterical outbreaks, but they may follow directly upon an injury or develop during the convalescence from an acute illness or be associated with disease of the generative organs.

Symptoms.—A useful division is into the convulsive and non-convulsive varieties.

Convulsive Hysteria.—(*a*) *Minor Forms.*—The attack, commonly following emotional disturbance, sets in suddenly or may be preceded by symptoms, called by the laity "hysterical," such as laughing and crying alternately, or a sensation of constriction in the neck, or of a ball rising in the throat—the *globus hystericus*. Sometimes, preceding the convulsive movements, there may be painful sensations arising from the pelvic, abdominal, or thoracic regions. From the description these sensations resemble auræ. They become more intense with the rising sensation of choking in the neck and difficulty in getting breath, and the patient falls into a more or less violent convulsion. The fall is not sudden, as in epilepsy, but the subject goes down, as a rule, easily, often picking a soft spot, like a sofa or an easy-chair, and in the movements apparently exercises care to do herself no injury. Yet at the same time she appears to be quite unconscious. The movements are clonic and disorderly, while the head and arms are thrown about in an irregular manner. The paroxysm after a few minutes slowly subsides, then the patient becomes emotional, and gradually regains consciousness. When questioned the patient may confess to having some knowledge of the events which have taken place, but, as a rule, has no accurate recollection. During the attack the abdomen may be much distended with flatus, and subsequently a large amount of clear urine may be passed. These attacks vary greatly in character. There may be scarcely any movements of the limbs, but after a nerve storm the patient sinks into a torpid, semi-unconscious condition, from which she is roused with great difficulty. In some cases from this state the patient passes into a condition of catalepsy.

(*b*) *Major Forms; Hystero-epilepsy.*—Typical instances passing through the various phases are very rare in the United States and in England. The attack is initiated by certain prodromata, chiefly minor hysterical manifestations, either foolish or unseemly behavior, excitement, sometimes dyspeptic symptoms with tympanites, or frequent micturition. Areas of hyperæsthesia may at this time be marked, the so-called hysterogenic spots so elaborately described by Richet. These are usually symmetrical and situated over the upper

dorsal vertebra, and in front in a series of symmetrically placed spots on the chest and abdomen, the most marked being those in the inguinal regions over the ovaries. Painful sensations or a feeling of oppression and a *globus* rising in the throat may be complained of prior to the onset of the convulsion, which, according to French writers, has four distinct stages: (1) Epileptoid condition, which closely simulates a true epileptic attack with tonic spasm (often leading to opisthotonos), grinding of the teeth, congestion of the face, followed by clonic convulsions, gradual relaxation, and coma. (2) Succeeding this is the period which Charcot has termed *clownism*, in which there is an emotional display and a remarkable series of contortions or of cataleptic poses. (3) Then in typical cases there is a stage in which the patient assumes certain attitudes expressive of the various passions—ecstasy, fear, beatitude, or erotism. (4) Finally consciousness returns and the patient enters upon a stage in which she may display very varied symptoms, chiefly manifestations of a delirium with the most extraordinary hallucinations. Visions are seen, voices heard, and conversations held with imaginary persons. In this stage patients will relate with the utmost solemnity imaginary events, and make extraordinary and serious charges against individuals. This sometimes gives a grave aspect to these seizures, for not only will the patient at this stage make and believe the statements, but when recovery is complete the hallucination sometimes persists. After an attack a patient may remain for days in a state of lethargy or trance.

Non-convulsive Forms.—So complex and varied is the clinical picture of hysteria that various manifestations are best considered according to the systems which are involved.

(*a*) *Disorders of Motion.*—(1) *Paralysis.*—These may be hemiplegic, paraplegic, or monoplegic. Hysterical diplegia is extremely rare. The paralysis either sets in abruptly or gradually, and may take weeks to attain its full development. *There is no type or form of organic paralysis which may not be simulated in hysteria.* Sensation is either lessened or lost on the affected side. The hysterical paraplegia is more common than hemiplegia. The loss of power is not absolute; the legs can usually be moved, but do not support the patient. The reflexes may be increased, though the knee-jerk is often normal. A spurious ankle clonus may sometimes be present. The feet are usually extended and turned inward in the equino-varus position. The muscles do not waste and the electrical reactions are normal. Other manifestations, such as paralysis of the bladder or aphonia, are usually associated with the hysterical paraplegia. Hysterical monoplegias may be facial, crural, or brachial. A condition of ataxia sometimes occurs with paresis. The incoördination may be a marked feature, and there are usually sensory manifestations.

(2) *Contractures and Spasms.*—The hysterical contractures may attack almost any group of voluntary muscles and be of the hemiplegic, paraplegic, or monoplegic type. They may come on suddenly or slowly, persist for months or years, and disappear rapidly. The contracture is most commonly seen in the arm, which is flexed at the elbow and wrist, while the fingers tightly grasp the thumb in the palm of the hand; more rarely the terminal phalanges are hyperextended as in athetosis. It may occur in one or in both legs, more commonly in one. The ankle clonus is present; the foot is inverted and the toes are strongly flexed. These cases may be mistaken for lateral sclerosis and the

difficulty in diagnosis may really be very great. The spastic gait is very typical, and with the exaggerated knee-jerk and ankle clonus the picture may be characteristic. Other forms of contracture may be in the muscles of the hip, shoulder, or neck; more rarely in those of the jaws—hysterical trismus—or in the tongue. Remarkable indeed are the local contractures in the diaphragm and abdominal muscles, producing a phantom tumor, in which just below and in the neighborhood of the umbilicus is a firm, apparently solid growth. According to Gowers, this is produced by relaxation of the recti and a spasmodic contraction of the diaphragm, together with inflation of the intestines with gas and an arching forward of the vertebral column. They are apt to occur in middle-aged women about the menopause, and are frequently associated with the symptoms of spurious pregnancy—*pseudo-cyesis*. The resemblance to a tumor may be striking, and I have known skilful diagnosticians to be deceived. The only safeguard is to be found in complete anæsthesia, when the tumor entirely disappears. Mitchell has reported an instance of a phantom tumor in the left pectoral region just above the breast, which was tender, hard, and dense.

Rhythmic Hysterical Spasm.—The movements may be of the arm, either flexion and extension, or, more rarely, pronation and supination. Clonic contractions of the sterno-cleido-mastoid or of the muscles of the jaws or of the rotatory muscles of the head may produce rhythmic movements of these parts. The spasm may be in one or both psoas muscles, lifting the leg in a rhythmic manner eight or ten times in a minute. In other instances the muscles of the trunk are affected, and every few moments there is a bowing movement—salaam convulsions—or the muscles of the back may contract, causing strong arching of the vertebral column and retraction of the head.

Tremor may be a purely hysterical manifestation, occurring either alone or with paralysis and contracture. It most commonly involves the hands and arms; more rarely the head and legs. The movements are small and quick. In the type described by Rendu the tremor may or may not persist during repose, but it is increased or provoked by volitional movements. Volitional or intentional tremor may exist, simulating closely the movements of insular sclerosis. Buzzard states that many instances of this disease in young girls are mistaken for hysteria.

(*b*) *Disorders of Sensation.—Anæsthesia* is most common, and usually confined to one half of the body. It may not be noticed by the patient. Usually it is accurately limited by the middle line and involves the mucous surfaces and deeper parts. The conjunctiva, however, is often spared. There may be hemianopia. This symptom may come on slowly or follow a convulsive attack. Sometimes the various sensations are dissociated and the anæsthesia may be only to pain and to touch. The skin of the affected side is usually pale and cool, and a pin-prick may not be followed by blood. With the loss of feeling there may be loss of muscular power. Curious trophic changes may be present, as in an interesting case of Weir Mitchell's, in which there was unilateral swelling of the hemiplegic side.

By metallotherapy, the application of certain metals, the anæsthesia or analgesia can be transferred to the other side of the body. It has been shown, however, that this phenomenon may be caused by the electro-magnet and by wood and various other agents, and is an effect of suggestion.

Hyperæsthesia.—Increased sensitiveness and pains occur in various parts of the body. One of the most frequent complaints is of pain in the head, usually over the sagittal suture, less frequently in the occiput. This is described as agonizing, and is compared to the driving of a nail into the part; hence the name *clavus hystericus*. Neuralgias are common. Hyperæsthetic areas, the hysterogenic points, exist on the skin of the thorax and abdomen, pressure upon which may cause minor manifestations or even a convulsive attack. Increased sensitiveness exists in the ovarian region, but is not peculiar to hysteria. Pain in the back is an almost constant complaint of hysterical patients. The sensitiveness may be limited to certain spinous processes, or it may be diffuse. In hysterical women the pains in the abdomen may simulate those of gastralgia and of gastric ulcer, or the condition may be almost identical with that of peritonitis; more rarely the abdominal pains closely resemble those of appendix disease.

Special Senses.—Disturbances of taste and smell are not uncommon and may cause a good deal of distress. Of ocular symptoms, retinal hyperæsthesia is the most common, and the patients always prefer to be in a darkened room. Retraction of the field of vision is common and usually follows a convulsive seizure. It may persist for years. The color perception may be normal even with complete anæsthesia. Hysterical deafness may be complete and may alternate or come on at the same time with hysterical blindness. Hysterical amaurosis may occur in children. One must carefully distinguish between functional loss of power and simulation.

(*c*) *Visceral Manifestations.—Respiratory Apparatus.*—Of disturbances in the respiratory rhythm, the most frequent, perhaps, is an exaggeration of the deeper breath, which is taken normally every fifth or sixth inspiration, or there may be a "catching" breathing, such as is seen when cold water is poured over a person. In hysterical dyspnœa there is no special distress and the pulse is normal. In what is known as the syndrome of Briquet there are shortness of breath, suppression of the voice, and paralysis of the diaphragm. The anhelation is extreme. In rare instances there is bradypnœa. Among laryngeal manifestations aphonia is frequent and may persist for months or even years without other special symptoms of the disease. Spasm of the muscles may occur with violent inspiratory efforts and great distress, and may even lead to cyanosis. Hiccough, or sounds resembling it, may be present for weeks or months at a time. Among the most remarkable of the respiratory manifestations are the hysterical cries. These may mimic the sounds produced by animals, such as barking, mewing, or grunting, and in France epidemics of them have been repeatedly observed. Extraordinary cries may be produced, either inspiratory or expiratory. Attacks of gaping, yawning, and sneezing may also occur.

The hysterical cough is a frequent symptom, particularly in young girls. It may occur in paroxysms, but is often a dry, persistent, croaking cough, extremely monotonous and unpleasant to hear. Sir Andrew Clark has called attention to a loud, barking cough (*cynobex hebetica*) occurring about the time of puberty, chiefly in boys belonging to neurotic families. The attacks, which last about a minute, recur frequently.

A peculiar form of hysterical hæmoptysis may be very deceptive and lead to the diagnosis of pulmonary disorders. The sputum is a pale-red fluid, not

so bright in color as in ordinary hæmoptysis, and on settling presents a reddish-brown sediment. It contains particles of food, pavement epithelium, red corpuscles, and micrococci, but no cylindrical or ciliated epithelium. It probably comes from the mouth or pharynx.

Digestive System.—Disturbed or depraved appetite, dyspepsia, and gastric pains are common in hysterical patients. The patient may have difficulty in swallowing the food, apparently from spasm of the gullet. There are instances in which the food seems to be expelled before it reaches the stomach. In other cases there is incessant gagging. In the hysterical vomiting the food is regurgitated without much effort and without nausea. This feature may persist for years without great disturbance of nutrition. The most striking and remarkable digestive disturbance in hysteria is the *anorexia nervosa* described by Sir William Gull. "To call it loss of appetite—anorexia—but feebly characterizes the symptom. It is rather an annihilation of appetite, so complete that it seems in some cases impossible ever to eat again. Out of it grows an antagonism to food which results at last and in its worst forms in spasm on the approach of food, and this in turn gives rise to some of those remarkable cases of survival for long periods without food" (Mitchell). There are three special features in anorexia nervosa: *First*, and most important, a psychical state, usually depressant, occasionally excited and restless. It is not always hysterical. *Secondly*, stomach symptoms, loss of appetite, regurgitation, vomiting, and the whole series of phenomena associated with nervous dyspepsia. *Thirdly*, emaciation, which reaches a grade seen only in cancer and dysentery. The patient finally takes to bed, and in extreme cases lies upon one side with the thighs and legs flexed, and contractures may occur. Food is either not taken at all or only upon urgent compulsion. The skin becomes wasted, dry, and covered with bran-like scales. No food may be taken for several weeks at a time, and attempts to feed may be followed by severe spasms. Although the condition looks so alarming, these cases, when removed from their home surroundings and treated by Weir Mitchell's method, sometimes recover in a remarkable way. It may take many months before any improvement is noted. Death, however, may follow with extreme emaciation. In a fatal case under my care the girl weighed only 49 pounds. No lesions were found post mortem.

Hysterical tympanites is a common feature, caused usually by tonic contraction of the diaphragm and retraction of the other abdominal muscles. It may be associated with the condition of peristaltic unrest (Kussmaul). Frequent discharges of fæces may be due to disturbance in either the small or large bowel. An obstinate form of diarrhœa is found in some hysterical patients, which proves very intractable and is associated especially with the taking of food. It seems an aggravated form of the looseness of bowels to which so many nervous people are subject on emotion or of the tendency which some have to diarrhœa immediately after eating. An entirely different form is that produced by what Mitchell calls the irritable rectum, in which scybala are passed frequently during the day, sometimes with great violence. Constipation is more frequent, however, and may be due to a loss of power in the muscles of the bowel, or in the abdominal muscles. In extreme cases the bowels may not be moved for two or three weeks, leading to great accumulation of fæces. Other disturbances are ano-spasm or intense pain in the rectum apart from any fissure. Hysterical ileus and fæcal vomiting are among the

most remarkable of hysterical phenomena. Following a shock there are constipation, tympanites, vomiting, sometimes hæmatemesis. The constipation grows worse, everything taken by the mouth is rejected, the vomitus becomes fæcal in character, even scybala are brought up, and suppositories and enemata are vomited. The symptoms may continue for weeks and then gradually subside. Laparotomy—even thrice in one patient—has shown a perfectly normal-looking condition of the bowels (Parkes Weber).

Cardio-vascular.—Rapid action of the heart on the slightest emotion, with or without the subjective sensation of palpitation, is often a source of great distress. A slow pulse is less frequent. Pains about the heart may simulate angina. Flushes in various parts are among the most common symptoms. Sweating may occur, or the *seborrhœa nigricans,* causing a darkening of the skin of the eyelids.

Among the more remarkable vaso-motor phenomena are the so-called stigmata or hæmorrhages in the skin, such as were present in the celebrated case of Louise Lateau. In many cases these are undoubtedly fraudulent, but if, as appears credible, such bleeding may exist in the hypnotic trance, there seems no reason to doubt its occurrence in the trance of prolonged religious ecstasy.

(*d*) *Joint Affections.*—To Sir Benjamin Brodie and Sir James Paget we owe the recognition of these extraordinary manifestations of hysteria. Perhaps no single affection has brought more discredit upon the profession, for the cases are very refractory, and finally fall into the hands of a charlatan or faith-healer, under whose touch the disease may disappear at once. Usually it affects the knee or the hip, and may follow a trifling injury. The joint is usually fixed, sensitive, and swollen. The surface may be cool, but sometimes the local temperature is increased. To the touch it is very sensitive and movement causes great pain. In protracted cases the muscles about the joint are somewhat wasted, and in consequence it looks larger. The pains are often nocturnal, at which time the local temperature may be much increased. While, as a rule, neuromimetic joints yield to proper management, there are interesting instances in the literature in which organic change has succeeded the functional disturbance.

Intermittent hydrarthrosis may be a manifestation of hysteria, occurring in the knee or other joints, sometimes with transient paresis.

(*e*) *Mental Symptoms.*—Mental perversions of all kinds are common in hysterical patients and not much dependence can be placed on statements either about themselves or about others. A morbid craving for sympathy may lead to the commission of all sorts of bizarre and foolish acts.

Hallucinations and delirium may alternate with emotional outbursts of an aggravated character. There is an interesting condition which may be spoken of as the *status hystericus.* For weeks or months they may be confined to bed, entirely oblivious to their surroundings, with a dilirium which may simulate that of delirium tremens, particularly in being associated with loathsome and unpleasant animals. The nutrition may be maintained, but there is always a very heavy, foul breath. With seclusion and care recovery usually takes place within three or four months. At the onset of these attacks and during convalescence the patients must be incessantly watched, as a suicidal tendency is by no means uncommon.

Of hysterical manifestations in the higher centres that of trance is the

most remarkable. This may develop spontaneously without any convulsive seizure, but more frequently it follows hysteroid attacks. Catalepsy may be present, a condition in which the limbs are plastic and remain in any position in which they are placed.

(*f*) *The Metabolism in Hysteria.*—In the ordinary forms of hysteria the urine does not show quantitative or qualitative changes, but in the severer types, characterized by convulsions, etc., there are important modifications; reduction in the urates and phosphates; the ratio of the earthy to the alkaline phosphates, normally 1:3, is 1:2, or even 1:1. The urine is also reduced in amount.

(*g*) *Self inflicted wounds,* more particularly burns, are sometimes met with in hysterical patients. I saw at the Hôtel Dieu, Paris, the remarkable condition described by Dieulafoy under the term Pathomimia, in which a young woman supposed to be the subject of a severe trophic disorder submitted to the amputation of the left arm before the confession was obtained that the lesions were self-inflicted! It is to be borne in mind that in Japan and other Eastern countries spontaneous tears occur in the soft parts, usually of the legs—the so-called Kamitachi disease, believed to be due to variations in atmospheric conditions, particularly during thunderstorms.

(*h*) *Hysterical Fever.*—In hysteria the temperature, as a rule, is normal. The cases with fever may be grouped as follows: (1) Instances in which the fever is the sole manifestation. These are rare, but I have seen cases in which the chronic course, the retention of the nutrition, and the entirely negative condition of the organs left no other diagnosis possible. In one case the patient had for four or five years an afternoon rise of temperature, reaching usually to 102° or 103°. She was well nourished and presented no pronounced hysterical symptoms, beyond a form of interrupted sighing respiration so often seen in hysteria. There was a marked neurotic history on one side of the family.

(2) Cases of hysterical fever with spurious local manifestations. These are very troublesome and deceptive cases. The patient may be suddenly taken ill with pain in various regions and elevation of temperature. The case may simulate meningitis. There may be pain in the head, vomiting, contracted pupils, and retraction of the neck—symptoms which may persist for weeks—and some anomalous manifestation during convalescence may alone indicate to the physician that he has had to deal with a case of hysteria, and has not, as he perhaps flattered himself, cured a case of meningitis. Mary Putnam Jacobi, in an article on hysterical fever, mentions a case in the service of Cornil which was admitted with dyspnœa, slight cyanosis, and a temperature of 39° C. The condition proved to be hysterical. There is also an hysterical pseudo-phthisis with pain in the chest, slight fever, and the expectoration of a blood-stained mucus. The cases of hysterical peritonitis may also show fever.

(3) *Hysterical Hyperpyrexia.*—It is a suggestive fact that the cases of paradoxical temperatures reported of late years, in which the thermometer has registered 112° to 120° or more, have been in women. Fraud has been practised in nearly all these cases.

Diagnosis.—Inquiry into the occurrence of previous manifestations and the mental conditions may give important information. These questions, as a rule, should not be asked the mother, who of all others is least likely to give

satisfactory information about the patient's condition. The occurrence of the globus hystericus, of emotional attacks, of weeping and crying is always suggestive. The points of difference between the convulsive attacks and true epilepsy were referred to in their description, and, as a rule, little difficulty is experienced in distinguishing between the two conditions. The hysterical paralyses are very variable and apt to be associated with anæsthesia. The contractures may at times be very deceptive, but the occurrence of areas of anæsthesia, of retraction of the visual field, and the development of minor hysterical manifestations give valuable indications. The contractures disappear under full anæsthesia. Special care must be taken not to confound the spastic paraplegia of hysteria with lateral sclerosis.

The visceral manifestations are usually recognized without much difficulty. The practitioner has constantly to bear in mind the strong tendency in hysterical patients to practice deception.

Treatment.—The prophylaxis in hysteria may be gathered from the remarks on the relation of education to the disease. The successful treatment of hysteria demands qualities possessed by few physicians. The first element is a due appreciation of the nature of the disease on the part of the physician and friends. It is pitiable to think of the misery which has been inflicted on these unhappy victims by the harsh and unjust treatment which has resulted from false views of the nature of the trouble; on the other hand, worry and ill health, often the wrecking of mind, body, and estate, are entailed upon the near relatives in the nursing of a protracted case of hysteria. The minor manifestations, attacks of the vapors, the crying and weeping spells, are not of much moment and rarely require treatment. The physical condition should be carefully looked into and the mode of life regulated so as to insure system and order in everything. A congenial occupation offers the best remedy for many of these manifestations. Any functional disturbance should be attended to and a course of tonics prescribed. Special attention should be paid to the action of the bowels.

Psychotherapy, in which the important features are hypnosis, suggestion, and reëducation.

Hypnosis.—The majority of hysterical patients can be hypnotized, but the general opinion now of those who know most on the subject is that by hypnosis alone hysteria is rarely cured. Sometimes a brilliant miracle is wrought in the case of hysterical paraplegia or hemiplegia, but as a routine treatment it has fallen into disfavor even in France.

Suggestion.—Babinski defines suggestion as "the action by which one endeavors to make another accept or realize an idea which is manifestly unreasonable." On the other hand, persuasion is applied when the ideas are reasonable, or at least are not in opposition to good sense. Most writers, however, use the word "suggestion" as meaning the introduction of mental associations and modifications of the patient's mental state leading to betterment. In proper hands it is a most powerful instrument, particularly when the patient has faith in the person who makes it. After a careful and sympathetic examination and testing the electrical reactions of the muscles of a paralyzed limb the suggestion to the hysteric, "Now I think you will be able to move it" may be all-sufficient. A strong, imperative command may sometimes have the same effect.

Reëducation.—In both hysteria and neurasthenia this should be the aim of all reasonable practice, but we must remember it is not always feasible: some of our patients would have to be rebuilt from the blastoderm. With patience and method much may be done, and the special merit of Weir Mitchell's work and of his system (which is not simply a rest cure, as many suppose) is that it is an elaborate plan of reëducation. The essentials are that the patient should be isolated from his friends and under the charge of an intelligent nurse. The physical condition is carefully studied and a rigid daily régime carried out: A milk diet of three to four quarts daily, rising to five or six, varying the food as the patient improves, and as the weight increases. This may be followed by a rapid gain in weight and the disappearance of all the unpleasant abdominal symptoms. Massage, hydrotherapy, and electricity are brought in as adjuncts, but very much depends upon the tact, patience, and, above all, the personality of the physician; the man counts more than the method. The mental condition has to be carefully studied and the patient's attitude toward life influenced by specially selected literature, careful conversation, and the suggestion of topics for thought.

The Analytical or Cathartic Method.—Introduced by Breuer and extended by Freud, it is in reality the old method of the confessional, in which the sinner poured out his soul in the sympathetic ear of the priest. It is a difficult procedure, not for all to attempt, exhausting alike to patient and doctor, and, when thoroughly carried out, time consuming. In the hands of those who have practised it, very good results have been obtained, particularly in young and carefully selected cases. The following statement of the method I take from Jelliffe ("System of Medicine," Vol. VII, page 866):

"His (Freud's) general procedure is to place the patient in a recumbent position, the physician sitting behind the patient's head at the end of the lounge. The physician thus remains practically out of sight of the patient, who is then asked to give a detailed account of his troubles, and to say everything that comes to the mind irrespective of its seeming logic or sense, and apart from disturbing, mortifying, or unnice suggestions. In all such histories gaps are inevitable. These the patient is urged to fill in by thinking closely of the attendant circumstances, speaking aloud all of the flitting thoughts that pass during this search ('free association'). All the thoughts are requested to be uttered, notwithstanding their disagreeable nature. The patient must exercise no critique and remain passive. It will be found that the disagreeable thoughts are pushed back with the greatest resistance. This is made all the more striking since the hysterical reaction, i. e., the symptom, is the symbolic expression of the realization of a repressed wish and gives the patient some gratification. A great effort is made to retain the symptom, especially as its origin is not really perceived, and since it represents, in symbol, the individual's former conscious strivings. In psycho-analysis one attempts to overcome all of these resistances, and by a series of judicious and tactful probings reconduct into the patient's consciousness the hidden thoughts which underlie these symptoms. Every symptom has some meaning; behind it there lies some associated mechanism, the origin of which the patient unconsciously or partly consciously represses. In the psycho-neurotic symbol may be read the cryptic expression of the original thought driven back and hidden.

"To slowly analyze and pick apart the mechanism is the object of the ana-

lytical method. One needs not only special tact for such excursions into the subtleties of the mental life of some individuals, but also a developed method of interpretation. Every act, every symbolic expression or action, lapse in speech, mannerism, needs to be carefully noted and its bearing coördinated. Freud lays particular emphasis on the analysis of dreams, since he believes that in the dream the subconscious, or the 'repressed conscious' is more apt to reveal itself. Hence a careful reading of Freud's 'Significance of Dreams' is of the greatest value in this study, also his 'Psychopathology of Every-day Life.' In his work on dreams he has developed to the full the chief directions along which his mind has traveled in the psychoanalytical method.

"It is of the utmost importance to trace back into the earliest years the striking emotional influences that have come into experience, as, for Freud, the hysterical reaction consists in a perverted type of reaction to these experiences. As is known, the blurring, or loss of an emotional influence—an affect, in short—is due to a number of factors. In normal life forgetting is the commonest type of a corrective adaptation, and forgetting is carried out with special ease if the emotional stress has not been excessive. Forgetting, however, is only a secondary phenomenon, and usually is more successful if the immediate reaction has been an adequate one. Such immediate reactions express themselves as tears, as anger, as impulsive acts, etc., and in such reactions the effect is discharged. In every-day life one calls it giving vent to one's feelings. If, however, the reaction is suppressed, the effect becomes united to the memory of the experience, and an emotional complex, or, to use a rather broad simile, a psychic boil, results, which must heal by absorption, by discharge, or by other means. Freud uses the term ab-react (abreagieren) to signify the adequate reaction, or discharge of such effects or their resulting complexes. Talking the whole thing over, giving vent to one's secrets and confessions are well-known abreactions.

"In hysteria certain of these complexes remain prominent; they are neither reacted too promptly, nor is their unpleasant feeling tone diminished by the blurring process of forgetting, although it is characteristic of the Freud point of view that the actual experience which gives rise to them becomes forgotten and the cause of the affect disturbance which becomes later converted, it may be into physical signs, remains apparently unknown to the patient. It must be dug out by psycho-analysis, and when once discovered catharsis takes place and the patient becomes cured."

Hydrotherapy is of great value, especially wet packs, salt baths, and various douches. General tonics, such as arsenic and iron, may be helpful, especially if the patients are nervous and anæmic. Sedatives are rarely indicated. Occasionally bromides may be necessary, but for the relief of sleeplessness all possible measures should be resorted to before the employment of drugs. The wet pack given hot or cold at night will usually suffice.

X. NEURASTHENIA

(*Psychasthenia*)

Definition.—A condition of weakness or exhaustion of the nervous system, giving rise to various forms of mental and bodily inefficiency.

The term, an old one, but first popularized by Beard, covers an ill-defined, motley group of symptoms, which may be either general and the expression of derangement of the entire system, or local, limited to certain organs; hence, the terms cerebral, spinal, cardiac, and gastric neurasthenia.

Etiology.—The causes may be grouped as hereditary and acquired.

(*a*) Hereditary.—We do not all start in life with the same amount of nerve capital. Parents who have led irrational lives, indulging in excesses of various kinds, or who have been the subjects of nervous complaints or of mental trouble, may transmit to their children an organization which is defective in what, for want of a better term, we must call "nerve force." Such individuals start handicapped with a neuropathic predisposition, and furnish a considerable proportion of our neurasthenic patients. As van Gieson sonorously puts it, "the potential energies of the higher constellations of their association centres have been squandered by their ancestors." So long as these individuals are content to transact a moderate business with their life capital, all may go well, but there is no reserve, and in the exigencies of modern life these small capitalists go under and come to us as bankrupts.

(*b*) Acquired.—The functions, though perverted most readily in persons who have inherited a feeble organization, may also be damaged in persons with no neuropathic predisposition by exercise which is excessive in proportion to the strength—i. e., by strain. The cares and anxieties attendant upon the gaining of a livelihood may be borne without distress, but in many persons the strain becomes excessive and is first manifested as *worry*. The individual loses the distinction between essentials and non-essentials, trifles cause annoyance, and the entire organism reacts with unnecessary readiness to slight stimuli, and is in a state which the older writers called irritable weakness. If such a condition be taken early and the patient given rest, the balance is quickly restored. In this group may be placed a large proportion of the neurasthenia which we see among business men, teachers, and journalists. Neurasthenia may follow the infectious diseases, particularly influenza, typhoid fever, and syphilis. The abuse of certain drugs, alcohol, tobacco, morphine may lead to a high grade of neurasthenia, though the drug habit is more often a result rather than a cause of the neurasthenia.

(*c*) Sexual Causes.—Undoubtedly the part played in the production of hysteria and allied neuroses by sexual factors is of the first importance. As already stated, Freud regards sexual trauma as the basis of hysteria, and he also regards neurasthenia as largely a product of disturbance in the sexual sphere. For him and his school the sexual impulses furnish the basis of the psychoneuroses. Repressed as they have to be in so many in our modern civilization, without normal outlet, the thought formations, retained in the unconscious state, express themselves by means of somatic phenomena—the objective features of hysteria and neurasthenia. *Cherchez la femme* is a safe rule in investigating a neurotic case. Freud may have ridden his hobby too hard, particularly in the insistence upon the importance of infantile sexuality, but in recognizing the rôle of the younger Aphrodite in the lives of men and women he has but followed the great master, Plato, who saw, while he deplored, the havoc wrought by her universal dominance.

The traumatic forms, especially those following upon railway accidents, will be separately considered.

Symptoms.—These are extremely varied, and may be general or localized; more often a combination of both. The appearance of the patient is suggestive, sometimes characteristic, but difficult to describe. Important information can be gained by the physician if he observes the patient closely as he enters the room—the way he is clothed, the manner in which he holds his body, his facial expression, and the humor which he is in. Loss of weight and slight anæmia may be present. The physical debility may reach a high grade and the patient may be confined to bed. Mentally the patients are usually low-spirited and despondent; women are frequently emotional.

The local symptoms may dominate the situation, and there have accordingly been described a whole series of types of the disease—cerebral, spinal, cardio-vascular, gastric, and sexual. In all forms there is a striking lack of accordance between the symptoms of which the patient complains and the objective changes discoverable by the physician. In nearly every clinical type of the disease the predominant symptoms are referable to pathological sensations and the psychic effects of these. Imperfect sleep is also complained of by a majority of patients, or, if not complained of, is found to exist on inquiry.

In the cerebral or psychic form the symptoms are chiefly connected with an inability to perform the ordinary mental work. Thus, a row of figures can not be correctly added, the dictation or the writing of a few letters is a source of the greatest worry, the transaction of petty details in business is a painful effort, and there is loss of power of fixed attention. With this condition there may be no headache, the appetite may be good, and the patient may sleep well. As a rule, however, there are sensations of fulness and weight or flushes, if not actual headache. Sleeplessness is a frequent concomitant of the cerebral form, and may be the first manifestation. Some of these patients are good-tempered and cheerful, but a majority are moody, irritable, and depressed.

Hyperæsthesia, especially to sensations of pain, is one of the main characteristics of almost all neurasthenic individuals. The sensations are nearly always referred to some special region of the body—the skin, eye muscles, the joints, the blood-vessels, or the viscera. It is frequently possible to localize a number of points painful to pressure (Valleix's points). In some patients there is marked vertigo, occasionally even resembling that of Ménière's disease.

If such pathological sensations continue for a long time the mood and character of the patient gradually alter. The so-called "irritable humor" develops. Many obnoxiously egoistic individuals met with in daily life are in reality examples of psychic neurasthenia. Everything is complained of. The patient demands the greatest consideration for his condition; he feels that he has been deeply insulted if his desires are not always immediately granted. He may at the same time have but little consideration for others. Indeed, in the severer forms of the disease he may show a malicious pleasure in attempting to make people who seem happier than himself uncomfortable. Such patients complain frequently that they are "misunderstood" by their fellows.

In many cases the so-called "anxiety conditions" gradually come on; one scarcely ever sees a case of advanced neurasthenia without the existence of some form of "anxiety." In the simpler forms of anxiety (nosophobic) there may be only a fear of impending insanity or of approaching death or of apo-

plexy. More frequently the anxious feeling is localized somewhere in the body —in the præcordial region, in the head, in the abdomen, in the thorax, or more rarely in the extremities.

In some cases the anxiety becomes intense and the patients are restless, and declare that they do not know what to do with themselves. They may throw themselves upon a bed, crying and complaining, and making convulsive movements with the hands and feet. Suicidal tendencies are not uncommon in such cases, and the patients may in desperation actually take their own lives.

Involuntary mental activity may be very troublesome; the patient complains that when he is overtired thoughts which he cannot stop or control run through his head with lightning-like rapidity. In other cases there is marked absence of ideas, the individual's mind being so filled up owing to the overexcitability of latent memory pictures that he is unable to form the proper associations for ideas called up by external stimuli. Sometimes a patient complains that a definite word, a name, a number, a melody, or a song keeps running in his head in spite of all he can do to abolish it.

In the severer cases the so-called "phobias" are common. The most frequent form perhaps is *agoraphobia*, in which patients the moment they come into an open space are oppressed by an exaggerated feeling of anxiety. They seem "frightened to death," and commence to tremble all over; they complain of compression of the thorax and palpitation of the heart. They may break into profuse perspiration and assert that they feel as though chained to the ground or that they can not move a step. It is remarkable that in some such cases the open space can be crossed if the individual be accompanied by some one, even by a child, or if he carry a stick or an umbrella! Other people are afraid to be left alone (monophobia), especially in a closed compartment (claustrophobia).

The fear of people and of society is known as anthropophobia. A whole series of other phobias have been described—batophobia, or the fear that high things will fall; pathophobia, or fear of disease; siderodromophobia, or fear of a railway journey; siderophobia or astrophobia, fear of thunder and lightning. Occasionally we meet with individuals who are afraid of everything and every one—victims of the so-called pantophobia.

The *special senses* may be disturbed, particularly vision. An aching or weariness of the eyeballs after reading a few minutes or flashes of light are common symptoms. The "irritable eye," the so-called nervous or neurasthenic asthenopia, is familiar to every family physician.

There may be acoustic disturbances—hyperalgesia and even true hyperacusia.

One of the most common of all the symptoms of neurasthenia is the *pressure in the head* complained of by these patients. This symptom, variously described, may be diffuse, but is more frequently referred to some one region—frontal, temporal, parietal, or occipital.

When the *spinal symptoms* predominate—spinal irritation or spinal neurasthenia—in addition to many of the features just mentioned, the patients complain of weariness on the least exertion, of weakness, pain in the back, intercostal neuralgiform pains, and of aching pains in the legs. There may be spots of local tenderness on the spine. The rachialgia may be spontaneous, or may be noticed only on pressure or movement. Occasionally there may be

disturbances of sensation, particularly a feeling of numbness and tingling, and the reflexes may be increased. Visceral neuralgias, especially in connection with the genital organs, are frequently met with. The aching pain in the back or in the back of the neck is the most constant complaint in these cases. In women it is often impossible to say whether this condition is one of neurasthenia or hysteria. It is in these cases that the disturbances of muscular activity are most pronounced, and in the French writings *amyosthenia* particularly plays an important rôle. The symptoms may be irritative or paretic, or a combination of both. Disturbances of coördination are not uncommon in the severer forms. These are particularly prone to involve the associated movements of the eye muscles, leading to asthenopic lack of accommodation. Drooping of one eyelid is very common, probably owing to insufficient innervation on the part of the sympathetic rather than to paresis of the oculomotor nerve. Occasionally Romberg's symptom may be present, and the patient, or even his physician, may fear a beginning tabes. More rarely there is disturbance of such finely coördinated acts as writing and articulation, not unlike those seen at the onset of general paresis. Such symptoms are always alarming, and the greatest care must be taken in establishing a diagnosis. That they may be the symptoms of pure neurasthenia, however, can no longer be doubted.

The reflexes in neurasthenia are usually increased, the deep reflexes especially never being absent. The condition of the superficial reflexes is less constant, though these, too, are usually increased. The pupils are often dilated, and the reflexes are usually normal. There may be inequality of the pupils in neurasthenia. Errors in refraction are common, the correction of which may give great relief.

In another type of cases the muscular weakness is extreme, and may go on even to complete motor helplessness. Very thorough examination is necessary before deciding as to the nature of the affection, since in some instances serious mistakes have been made. Here belong the *atremia* of Neftel, the *akinesia algera* of Möbius, and the neurasthenic form of *astasia abasia* described by Binswanger.

In other cases the *cardio-vascular* symptoms are the most distressing, and may occur with only slight disturbance of the cerebro-spinal functions, though the conditions are nearly always combined. Palpitation of the heart, irregular and very rapid action (neurasthenic tachycardia), and pains and oppressive feelings in the cardiac region are the most common symptoms. The slightest excitement may be followed by increased action of the heart, sometimes associated with sensations of dizziness and anxiety, and the patients frequently have the idea that they suffer from serious disease of this organ. Attacks of pseudo-angina may occur.

Vaso-motor disturbances constitute a special feature of many cases. Flushes of heat, especially in the head, and transient hyperæmia of the skin may be very distressing symptoms. Profuse sweating may occur, either local or general, and sometimes nocturnal. The pulse may show interesting features, owing to the extreme relaxation of the peripheral arterioles. The arterial throbbing may be everywhere visible, almost as much as in aortic insufficiency. The pulse, too, may under these circumstances have a somewhat water-hammer quality. The capillary pulse may be seen in the nails, on the lips, or on the

Sometimes used to "drop" heart which may be dicated

margins of a line drawn upon the forehead, and I have on several occasions seen pulsation in the veins of the back of the hand. A characteristic symptom in some cases is the *throbbing aorta*. This "preternatural pulsation in the epigastrium," as Allan Burns calls it, may be extremely forcible and suggest the existence of abdominal aneurism. The subjective sensations associated with it may be very unpleasant, particularly when the stomach is empty.

In women especially, and sometimes in men, the peripheral blood-vessels are contracted, the extremities are cold, the nose is red or blue, and the face has a pinched expression. These patients feel much more comfortable when the cutaneous vessels are distended, and resort to various means to favor this (wearing of heavy clothing, use of diffusible stimulants).

The general features of *gastro-intestinal neurasthenia* have been dealt with under the section of nervous dyspepsia. The connection of these cases with dilatation of the stomach, floating kidney, and the condition which Glénard calls *enteroptosis* has already been mentioned.

Sexual neurasthenia is a condition in which there is an irritable weakness of the sexual organs manifested by nocturnal emissions, unusual depression after intercourse, and often by a distressing dread of impotence. The mental condition of these patients is most pitiable, and they fall an easy prey to quacks and charlatans of all kinds. In males these symptoms are frequently due to diseased conditions in the deep urethra, especially of the verumontanum, and prostate.

Spermatorrhœa is the bugbear of the majority. They complain of continued losses, usually without accompanying pleasurable sensations. After defecation or micturition there may be seminal discharges. Microscopic examination sometimes reveals the presence of spermatozoa. Actual nervous impotence is not uncommon. The "painful testicle" is a well-known neurasthenic phenomenon. In the severer cases, especially those bearing the stigmata of degeneration, there may be evidence of sexual perversion.

In females it is common to find a tender ovary, and painful or irregular menstruation.

In all forms of neurasthenia the condition of the urine is important. Many cases are complicated with the symptoms of the condition known as lithæmia, and so marked may this be that some have indeed made a special form of lithæmic neurasthenia. Polyuria may be present, but is more common in hysteria. With disturbed digestion the urates and oxalates may be in excess.

Diagnosis.—*Psychasthenia.*—Under this term Janet would separate from neurasthenia the cases characterized by mental, emotional, and physical disturbances, imperative ideas, phobias of all sorts, doubts, enfeebled will, uncontrollable movements, and many of the borderland features of the insanity of young persons. It is really an inherited psychoneurosis, while neurasthenia is usually acquired. Obsessions of all sorts characterize the condition and there may be a feeling of unreality and even of loss of personality. How complicated the condition may be is shown from the following varieties distinguished by Janet: (1) The *doubter,* in whom obsessive ideas are not very precise, more of the nature of a general indication rather than a specific idea, such as a craze for research, for explanation, for computing. (2) The *scrupulous,* whose obsessions are of a moral nature. Their manias are of literal-

ness of statement, of exact truth, of conjuration, of reparation, of symbols, etc. (3) The *criminal*, whose obsessive ideas are of homicide, theft, and other overt acts. The impulsive idea is stronger in this than in the other varieties. (4) The *inebriates*, dipsomaniac, morphinomaniac, etc., in whom the impulse seems to be least resistible. (5) The *genesically perverted*. (6) *Delirious psychasthenia*, a condition in which a delirious state of mind occurs, connected with the obsession.

The anxiety conditions and various phobias, as well as the different varieties of tic and the occupation neuroses when they accompany neurasthenia, are regarded as complications dependent in the majority of instances upon faulty heredity.

Neurasthenia is a disease above all others which has to be diagnosed from the subjective statements of the patient, and from an observation of his general behavior rather than from the physical examination. The physical examination is of the highest importance in excluding other diseases likely to be confounded with it. That somatic changes occur and that physical signs are often to be made out is very true, but there is nothing typical or pathognomonic in these objective changes.

The hypochondriac differs from the neurasthenic in the excessive psychic distortion of the pathological sensations to which he is subject. He is the victim of actual delusions regarding his condition.

The confusion of neurasthenia with hysteria is still more frequent; in women especially a diagnosis of hysteria is often made when in reality the condition is one of neurasthenia. In the absence of hysterical paroxysms, of crises, and of those marked emotional and intellectual characteristics of the hysterical individual the diagnosis of hysteria should not be made. Of course, in many of the cases of hysteria definite hysterical stigmata (hysterical paralyses, convulsions, contractures, anæsthesias, alterations in the visual field, etc.) are present, and the diagnosis is not difficult.

Epilepsy is not likely to be confounded with neurasthenia if there be definite epileptic attacks, but the cases of *petit mal* may be puzzling.

The onset of exophthalmic goitre may be mistaken for neurasthenia, especially if there be no exophthalmos at the beginning. The emotional disturbances and the irritability of the heart may mislead the physician. In pronounced cases of nervous prostration the differential diagnosis from the various psychoses may be extremely difficult.

The two forms of organic disease of the nervous system with which neurasthenia is most likely to be confounded are tabes and general paresis. The symptoms of the spinal form of neurasthenia may resemble those of the former disease, while the symptoms of the psychic or cerebral form of neurasthenia may be very similar to those of general paresis. The diagnosis, as a rule, presents no difficulty if the physician be careful to make a thorough routine examination. It is only the superficial study of a case that is likely to lead one astray. In tabes especially a consideration of the sensory disturbances, of the deep reflexes, and of the pupillary findings will always establish the presence or absence of the disease. In general paresis there is sometimes more difficulty. The onset of general paresis is often characterized by the appearance of symptoms quite like those of ordinary neurasthenia, and the family physician may entirely overlook the grave nature of the malady. The mis-

take in the other direction is, however, perhaps just as common. A physician who once or twice has seen a case of general paresis arise out of what appeared to be one of pronounced neurasthenia is too prone afterward to suspect every neurasthenic to be developing the malign affection. The most marked symptoms, however, of psychic exhaustion do not justify a diagnosis of general paresis even when the history is suspicious, unless along with it there is a definite paresis of the pupils, of the facial muscles, or of the muscles of articulation. A history of syphilis or of chronic alcoholism or morphinism associated with severe psychic exhaustion should, of course, put one always on his guard, and the physician should be sharply on the lookout for the appearance of intellectual defects, paraphasia, facial paresis, and sluggishness of the pupils.

Treatment.—PROPHYLAXIS.—Many patients come under our care a generation too late for satisfactory treatment, and it may be impossible to restore the exhausted capital. The greatest care should be taken in the rearing of children of neuropathic predisposition. From a very early age they should be submitted to a process of "psychic hardening," every effort being made to strengthen the bodily and mental condition. Even in infancy the child should not be pampered. Later on the greatest care should be exercised with regard to food, sleep, and school work. Complaints of children should not be too seriously considered.

Much depends upon the example set by the parents. A restless, emotional, constantly complaining mother will rack the nervous system of a delicate child. In some instances, for the welfare of a developing boy or girl, the physician may find it necessary to advise its removal from home.

Neurotic children are especially liable during development to fits of temper and of emotional disturbance. These should not be too lightly considered. Above all, violent chastisement in such cases is to be avoided, and loss of temper on the part of the parent or teacher is particularly pernicious for the nervous system of the child. Where possible, in such instances, the best treatment is to put the obstreperous child immediately to bed, and if the excitement and temper continue a warm bath followed by a cool douche may be effective. If he be put to bed after the bath sleep soon follows.

Special attention is necessary at puberty in both boys and girls. If there be at this period any marked tendency to emotional disturbance or to intellectual weakness the child should be removed from school and every care taken to avoid unfavorable influences.

PERSONAL HYGIENE.—Throughout life individuals of neuropathic predisposition should obey scrupulously certain hygienic and prophylactic rules. Intellectual work especially should be judiciously limited and should alternate frequently with periods of repose. Excitement of all kinds should of course be avoided, and such individuals will do well to be abstemious in the use of tobacco, tea, coffee, and alcohol, if, indeed, they be permitted to use these substances at all. The habit, happily becoming very common, of taking at least once a year a prolonged holiday away from the ordinary environment, in the woods, in the mountains, or at the seashore, should be urgently enjoined upon every neuropathic individual. In many instances it is found to be the greatest relief and rest if the patient can take his holiday away from his relatives.

During ordinary life nervous people should, during some portion of each

day, pay rational attention to the body. Cold baths, swimming, exercises in the gymnasium, gardening, golf, lawn tennis, cricket, hunting, shooting, rowing, sailing, and bicycling are of value in maintaining the general nutrition. Such exercises are, of course, to be recommended only to individuals physically equal to them. If neurasthenia be once well established the greatest care must be observed in the ordering of exercise. Many nervous girls have been completely broken down by following injudicious advice with regard to long walks.

Treatment of the Condition.—The treatment of neurasthenia when once established presents a varied problem to the thoughtful physician. Every case must be handled upon its own merits, no two, as a rule, requiring exactly the same methods. In general it will be the aim of the medical adviser to remove the patient as far as possible from the influences which have led to his downfall, and to restore to normal the nervous mechanisms which have been weakened by injurious influences. The general character of the individual, his physical and social status, must of course be considered and the therapeutic measures carefully adjusted to these.

The diagnosis having been settled, the physician may assure the patient that with prolonged treatment, during which his coöperation with the physician is absolutely essential, he may expect to get well. He must be told that much depends upon himself and that he must make a vigorous effort to overcome certain of his tendencies, and that all his strength of will will be needed to further the progress of the cure. In the case of business or professional men, in whom the condition develops as a result of overwork or overstudy, it may be sufficient to enjoin absolute rest with change of scene and diet. A trip abroad, with a residence for a month or two in Switzerland, or, if there are symptoms of nervous dyspepsia, a residence at one of the Spas will usually prove sufficient. The excitement of the large cities abroad should be avoided. The longer the disease has lasted and the more intense the symptoms have been, the longer the time necessary for the restoration of health. In cases of any severity the patient must be told that at least six months' complete absence from business, under strict medical guidance, will be necessary. Shorter periods may of course be of benefit, which, however, as a rule, will be only temporary.

It will often be found advisable to make out a daily programme, which shall occupy almost the whole time of the patient. At first he need know nothing about this, the case being given over entirely to the nurse. As improvement advances, moderate physical and intellectual exercises, alternating frequently with rest and the administration of food, may be undertaken. Some one hour of the day may be left free for reading, correspondence, conversation, and games. In some instances the writing of letters is particularly harmful to the patient and must be prohibited or limited. Cultured individuals may find benefit from attention to drawing, painting, modelling, translating from a foreign language, the making of abstracts, etc., for short periods in the day.

In not a few cases, including a large proportion of neurasthenic women, a systematic Weir Mitchell treatment rigidly carried out should be tried. The patient must be isolated from his friends, and any regulations undertaken must be strictly adhered to, the consent of the patient and his family having first been gained. The treatment of the gastric and intestinal symptoms so im-

portant in this condition has already been considered. For the irregular pains, particularly in the back and neck, the thermo-cautery is invaluable.

Hydrotherapy is indicated in nearly every case if it can be properly applied. Much can be done at home or in an ordinary hospital, but for systematic hydrotherapeutic treatment residence in a suitable sanitarium is necessary. I have found the wet pack of especial value. Particularly at night, in cases of sleeplessness, it is perhaps the best remedy against insomnia we have. Salt baths are more helpful to some patients. The various forms of douches, partial packs, foot baths, etc., may be valuable in individual cases. Electrotherapy is of some value, though only in combination with psychic treatment and hydrotherapy.

Special care should be given to the recognition of local disease and proper measures instituted. Attention to the eyes is important. Infection of the naso-pharynx, sinus disease, visceroptosis, or anæmia should be corrected. In women the pelvic organs and in men the deep urethra and prostate may require treatment.

Treatment by drugs should be avoided as much as possible. They are of benefit chiefly in the combating of single symptoms. Alcohol, morphia, chloral, or cocaine should never be given. General tonics may be helpful, especially if the individual be anæmic. Arsenic and more often iron are then indicated. For the severer pains and nervous attacks some sedative may occasionally be necessary, especially at the beginning of the treatment. The bromides may here be given with advantage. An occasional dose of phenacetin or aspirin may be required, but the less of these substances we can get along with the better. For the relief of sleeplessness all possible measures should be resorted to before the employment of drugs. The wet pack will usually suffice. If absolutely necessary to give a drug, sulphonal, trional, or amylene hydrate may be employed.

In cases in which the anxiety conditions are disturbing the cautious use of opium in pill form may be necessary, since, as in the psychoses, opium here will sometimes yield permanent relief. A prolonged treatment with opium is, however, never necessary in neurasthenia.

Psychotherapy.—Hypnotism is rarely indicated. Carefully practiced suggestion is most helpful and the psycho-analytic method of Freud, as described under Hysteria, may be tried.

The use of religious ideas and practices may be most helpful, and this has come into vogue in various forms, as Christian Science, Emmanuelism, Mental Healing, etc. It is an old story. In all ages, and in all lands, the prayer of faith, to use the words of St. James, has healed the sick; and we must remember that amid the Æsculapian cult, the most elaborate and beautiful system of faith healing the world has seen, scientific medicine took its rise. As a profession, consciously or unconsciously, more often the latter, *faith* has been one of our most valuable assets, and Galen expressed a great truth when he said, "He cures most successfully in whom the people have the greatest confidence." It is in these cases of neurasthenia and psychasthenia, the weak brothers and the weak sisters, that the personal character of the physician comes into play, and once let him gain the confidence of the patient, he can work just the same sort of miracles as Our Lady of Lourdes or Ste. Anne de Beaupré. Three elements are necessary: first, a strong personality in whom the individual has

faith—Christ, Buddha, Æsculapius (in the days of Greece), one of the saints, or, what has served the turn of common humanity very well, a physician. Secondly, certain accessories—a shrine, a sanctuary, the services of a temple, or for us a hospital or its equivalent, with a skillful nurse. Thirdly, suggestion, either of the "only believe," "feel it," "will it" attitude of mind, which is the essence of every cult and creed, or of the active belief in the assurance of the physician that the precious boon of health is within reach.

XI. THE TRAUMATIC NEUROSES

(*Railway Brain and Railway Spine; Traumatic Hysteria*)

Definition.—A morbid condition following shock which presents the symptoms of neurasthenia or hysteria or of both. The condition is known as "railway-brain" and "railway spine."

Erichsen regarded the condition as the result of inflammation of the meninges and cord, and gave it the name railway spine. Walton and J. J. Putnam, of Boston, were the first to recognize the hysterical nature of many of the cases, and to Westphal's pupils we owe the name traumatic neurosis.

Etiology.—The condition follows an accident, often in a railway train, in which injury has been sustained, or succeeds a shock or concussion, from which the patient may apparently not have suffered in his body. A man may appear perfectly well for several days, or even a week or more, and then develop the symptoms of the neurosis. Bodily shock or concussion is not necessary. The affection may follow a profound mental impression; thus, an engine-driver ran over a child, and received thereby a very severe shock, subsequent to which the most pronounced symptoms of neurasthenia developed. Severe mental strain combined with bodily exposure may cause it, as in a case of a naval officer who was wrecked in a violent storm and exposed for more than a day in the rigging before he was rescued. A slight blow, a fall from a carriage or on the stairs may suffice.

Symptoms.—The cases may be divided into three groups: simple neurasthenia, cases with marked hysterical manifestations, and cases with severe symptoms indicating or simulating organic disease.

(*a*) Simple Traumatic Neurasthenia.—The first symptoms usually develop a few weeks after the accident, which may or may not have been associated with an actual trauma. The patient complains of headache and tired feelings. He is sleepless and finds himself unable to concentrate his attention properly upon his work. A condition of nervous irritability develops, which may have a host of trivial manifestations, and the entire mental attitude of the person may for a time be changed. He dwells constantly upon his condition, gets very despondent and low-spirited, and in extreme cases melancholia may develop. He may complain of numbness and tingling in the extremities, and in some cases of much pain in the back. The bodily functions may be well performed, though such patients usually have, for a time at least, disturbed digestion and loss in weight. The physical examination may be entirely negative. The reflexes are slightly increased, as in ordinary neurasthenia. The pupils may be unequal; the cardio-vascular changes already described in

neurasthenia may be present in a marked degree. According as the symptoms are more spinal or more cerebral, the condition is known as railway brain or railway spine.

(2) Cases with Marked Hysterical Features.—Following an injury of any sort, neurasthenic symptoms, like those described above, may develop, and in addition symptoms regarded as characteristic of hysteria. The emotional element is prominent, and there is but slight control over the feelings. The patients have headache, backache, and vertigo. A violent tremor may be present, and, indeed, constitutes the most striking feature of the case. In the case of an engineer who developed subsequent to an accident a series of nervous phenomenon the most marked feature was an excessive tremor of the entire body, which was specially manifest during emotional excitement. The most pronounced hysterical symptoms are the sensory disturbances. As first noted by Putnam and Walton, hemianæsthesia may occur as a consequence of traumatism. This is a common symptom in France, but rare in England and in the United States. Achromatopsia may exist on the anæsthetic side. A second, more common, manifestation is limitation of the field of vision, similar to that which occurs in hysteria.

(3) Cases in Which the Symptoms Suggest Organic Disease of the Brain and Cord.—As a result of spinal concussion, without fracture or external injury, there may subsequently develop symptoms suggestive of organic disease, which may come on rapidly or at a late date. In a case reported by Leyden the symptoms following the concussion were at first slight and the patient was regarded as a simulator, but finally the condition became aggravated and death resulted. The post mortem showed a chronic pachymeningitis, which had doubtless resulted from the accident. The cases in this group about which there is so much discussion are those which display marked sensory and motor changes. Following an accident in which the patient has not received external injury a condition of excitement may develop within a week or ten days; he complains of headache and backache, and on examination sensory disturbances are found, either hemianæsthesia or areas on the skin in which the sensation is much benumbed; or painful and tactile impressions may be distinctly felt in certain regions, and the temperature sense is absent. The distribution may be bilateral and symmetrical in limited regions or hemiplegic in type. Limitation of the field of vision is usually marked in these cases, and there may be disturbance of the senses of taste and smell. The superficial reflexes may be diminished; usually the deep reflexes are exaggerated. The pupils may be unequal; the motor disturbances are variable. The French writers describe cases of monoplegia with or without contracture, symptoms upon which Charcot lays great stress as a manifestation of profound hysteria. The combination of sensory disturbances—anæsthesia or hyperæsthesia—with paralysis, particularly if monoplegic, and the occurrence of contractures without atrophy and with normal electrical reactions, may be regarded as distinctive of hysteria.

In rare cases following trauma and succeeding to symptoms which may have been regarded as neurasthenic or hysterical there are organic changes which may prove fatal. That this sequence occurs is demonstrated clearly by recent post mortem examinations. The features upon which the greatest reliance can be placed as indicating organic change are optic atrophy, bladder

symptoms, particularly in combination with tremor, paresis, and exaggerated reflexes.

The anatomical changes in this condition have not been very definite. When death follows spinal concussion within a few days there may be no apparent lesion, but in some instances the brain or cord has shown punctiform hæmorrhages. Edes has reported 4 cases in which a gradual degeneration in the pyramidal tracts followed concussion or injury of the spine; but in all these cases there was marked tremor and the spinal symptoms developed early, or followed immediately upon the accident.

Diagnosis.—A condition of fright and excitement following an accident may persist for days or even weeks, and then gradually pass away. The symptoms of neurasthenia or of hysteria which subsequently develop present nothing peculiar and are identical with those which occur under other circumstances. Care must be taken to recognize simulation, and, as in these cases the condition is largely subjective, this is sometimes extremely difficult. In a careful examination a simulator will often reveal himself by exaggeration of certain symptoms, particularly sensitiveness of the spine, and by increasing voluntarily the reflexes. Maunkopff suggests as a good test to take the pulse rate before, during, and after pressure upon an area said to be painful. If the rate is quickened, it is held to be proof that the pain is real. This is not, however, always the case. It may require a careful study of the case to determine whether the individual is honestly suffering from the symptoms of which he complains. A still more important question is, Has the patient organic disease? The symptoms given under the first two groups of cases may exist in a marked degree and may persist for several years without the slightest evidence of organic change. Hemianæsthesia, limitation of the field of vision, monoplegia with contracture, may all be present as hysterical manifestations, from which recovery may be complete. In our present knowledge the diagnosis of an organic lesion should be limited to those cases in which optic atrophy, bladder troubles, and signs of sclerosis of the cord are well marked—indications either of degeneration of the lateral columns or of multiple sclerosis. Examination by the X-rays is an important aid and has showed in some cases definite injury to the spine.

Prognosis.—A majority of patients with traumatic hysteria recover. In railway cases, so long as litigation is pending and the patient is in the hands of lawyers, the symptoms usually persist. Settlement is often the starting-point of a speedy and perfect recovery. On the other hand, there are a few cases in which the symptoms persist even after the litigation has been closed; the patient goes from bad to worse and psychoses develop, such as melancholia, dementia, or occasionally progressive paresis. And, lastly, in extremely rare cases organic lesions may occur as a sequence of the traumatic neurosis.

The function of the physician acting as medical expert in these cases consists in determining (*a*) the existence of actual disease, and (*b*) its character, whether simple neurasthenia, severe hysteria, or an organic lesion. The outlook for ultimate recovery is good except in cases which present the more serious symptoms above mentioned. Nevertheless it must be borne in mind that traumatic hysteria is one of the most intractable affections which we are called upon to treat. In the treatment of the traumatic neuroses the practi-

tioner may be guided by the principles laid down in the preceding chapter, in which the treatment of neurasthenia in general has been described.

XII. OTHER FORMS OF FUNCTIONAL PARALYSIS

Periodical Paralysis.—The periodical paralysis of the ocular muscles, which may recur for years, has already been referred to. A periodical paralysis involving the general muscles, also a "family" affection, may return with great regularity. Goldflam described twelve cases in one family, the heredity being through the mother. In the United States E. W. Taylor described eleven cases in one family in five generations. Holtzapple, of York, Pa., reports 16 cases in one family. Six of the number died in an attack.

The clinical picture is similar in all recorded cases. The paralysis involves, as a rule, the arms and legs, but may be general below the neck. It comes on in healthy persons without apparent cause, and often during sleep. At first there may be weakness of the limbs, a feeling of weariness and sleepiness, but rarely sensory symptoms. The paralysis, beginning in the legs, to which it may be confined, is usually complete within the first twenty four hours. The neck muscles are sometimes involved, and occasionally those of the tongue and pharynx. The cerebral nerves and the special senses are, as a rule, unaffected. The temperature is normal or subnormal and the pulse slow. The deep reflexes are diminished, sometimes abolished, and the skin reflexes may be enfeebled. A most remarkable feature is the extraordinary reduction or complete abolition of the faradic excitability of both muscles and nerves.

Improvement begins within a few hours or a day or two, the paralysis disappearing completely and the patient becoming perfectly well. The attacks usually recur at intervals of one to two weeks, but they may return daily. They generally cease after the fiftieth year. There may be signs of acute dilatation of the heart during the attack. In the three cases reported by J. K. Mitchell, Flexner, and Edsall a diminished kreatinin excretion for several days before and at the beginning of a seizure was repeatedly found. There was a rise to normal after the attacks. Potassium citrate in full doses either shortened or aborted the paralyses.

Astasia; Abasia.—These terms, indicating respectively inability to stand and inability to walk, have been applied by Charcot and Blocq to diseased conditions characterized by loss of the power of standing or of walking, with retention of muscular power, coördination, and sensation. Blocq's definition is as follows: "A morbid state in which the impossibility of standing erect and walking normally is in contrast with the integrity of sensation, of muscular strength, and of the coördination of the other movements of the lower extremities." The condition forms a symptom group, not a morbid entity, and is probably a functional neurosis. Knapp analyzed 50 cases, of which half were in women. In 21 cases hysteria was present; in 3, chorea; in 2, epilepsy; and in 4, intention psychoses. As a rule, the patients, though able to move the feet and legs perfectly when in bed, are either unable to walk properly or can not stand at all. The disturbances have been very varied, and different forms have been recognized. The commonest, according to Knapp's analysis of the recorded cases, is the paralytic, in which the legs give out as

the patient attempts to walk and "bend under him as if made of cotton." "There is no rigidity, no spasm, no incoördination. In bed, sitting, or even while suspended, the muscular strength is found to be good." Other cases are associated with spasm or ataxia; thus there may be movements which stiffen the legs and give to the gait a somewhat spastic character. In other instances there are sudden flexions of the legs, or even of the arms, or a saltatory, spring-like spasm. In a majority of the cases it is a manifestation of a neurosis allied to hysteria.

The cases, as a rule, recover, particularly in young persons. Relapses are not uncommon. The rest treatment and static electricity should be employed.

H. VASO-MOTOR AND TROPHIC DISORDERS

I. RAYNAUD'S DISEASE

Definition.—A vascular change, without organic disease of the vessels, chiefly seen in the extremities, but occurring also in the internal parts, in which a persistent ischæmia or a passive hyperæmia leads to disturbance of function or to loss of vitality with necrosis.

Etiology.—It is a comparatively rare disease. There were only 19 cases in about 20,000 medical patients admitted to the Johns Hopkins Hospital. Women are more frequently attacked than men—62.5 to 37.5 per cent. in Monro's series.

Sixty per cent. of the cases occurred in the second and third decades, but no age is exempt. A case has been reported in a six-months-old child and in a woman of 77 years.

Several members of a family may be affected. Neurotic and hysterical patients are more prone to the disease. Damp and cold weather, ~~as in Great Britain,~~ appears to favor its occurrence. Severe chilblain leading to superficial necrosis represents a type of the malady. In the infectious diseases areas of multiple necrosis occur, but, as a rule, the distribution is very different, and such cases should not be included under Raynaud's disease, nor should the local gangrene associated with arteritis.

Pathology.—According to the definition, cases are excluded in which organic disease of the vessels is present. In advanced cases sclerosis of the blood-vessels has been found; and neuritis has been described, but neither is an essential factor. Changes in the spinal cord have been reported, but in a majority of all cases the examination has been negative. The local syncope is an expression of a widespread constrictor influence causing spasm of the arteries and arterioles, so that not a drop of blood enters a part. This may be followed in an hour or two, or less, by active hyperæmia; the arteries and arterioles dilate widely and the dead-white finger becomes a bright pink. While hyperæmia may follow the ischæmia directly, more commonly there is an intervening period of asphyxia in which the finger becomes blue. In frost-bite, active hyperæmia, cyanosis, and local syncope is the order. In Raynaud's disease the order is usually syncope, asphyxia, and hyperæmia. In frost-bite it seems clear that the asphyxia is due to a backward flow from the veins, to which the local syncope yields as the part thaws, before the arteries

passing to the part can be felt to pulsate. The asphyxia of Raynaud's disease may be due to the same cause; contraction of the veins has been seen by Barlow and by Weiss, but that was when the asphyxia already existed. The first thing must be the relaxation of the spasm of the venules and veins to permit of the blood entering the empty capillaries. In moderate grades of asphyxia some little blood trickles through the sluice gates, but in the deep purple skin of a typical example of Raynaud's disease the circulation has ceased and death of the part is imminent. The necrosis is a simple matter, as simple as if a string is tied tightly about the finger-tip.

The disease is the result of some as yet unknown instability of the vaso-motor centres.

Symptoms.—There are various grades of the disease, of which mild, moderate, and severe types may be recognized. In the mild forms the disease never gets beyond the stage of such vascular disturbance as is frequently seen in chilblains. The hands alone may be affected, ~~more often the hands and feet.~~ In the winter, on the slightest exposure, there is acro-cyanosis, which gives place in the warmth to active hyperæmia, sometimes with swelling, throbbing, and aching. The so-called "beefsteak" hand is often a great annoyance to women. It is a vaso-motor disturbance representing a potential case of Raynaud's disease. In these mild attacks ~~I have seen~~ one finger white and the adjacent ones red and blue.

The condition may persist for years and never pass on to necrosis. In a case of moderate severity a woman, aged say twenty or twenty five, after a period of worry or ill health, has pains in the fingers, or a numbness or tingling; then she notices that they are white and cold, and in an hour or so they become red and hot. Within a day or two a change occurs; they remain permanently blue perhaps as far as the second joint or to the knuckles. There is pain, sometimes severe enough to require morphia. The cyanosis persists and the tip of one finger or the terminal joint of another gets darker and a few blebs form. The other fingers show signs of restored circulation, but necrosis has occurred in the pad of one finger and perhaps the terminal inch of another. The necrotic parts gradually separate, and the patient may never have another attack, or in a year or two there is a recurrence.

The severer form is a terrible malady, and may affect fingers and toes at once and with them sometimes the tip of the nose and the ears. The pain is of great severity. Both feet may be swollen to the ankle with the toes black. It may look as if both feet would become gangrenous, but as a rule the process subsides, and in a case even of great severity only the tips of the toes are lost. A severe attack of this sort may last three or four months, when the patient recovers with the loss of two or three fingers or toes, a snip off the edge of both ears and a scar on the tip of the nose. Attacks of this severity may occur year by year, and there are terrible instances in which the patients have lost both hands and feet.

Of the parts affected Monro states that in 43 per cent. of the cases one or both of the upper extremities is involved. Parts other than the extremities may be attacked, as the chin, lips, nates, and eyelids.

Complications.—Temporary amblyopia due to spasm of the retinal vessels, transient aphasia, and transient hemiplegia have been met with. In a case ~~which I have~~ reported there were three attacks of aphasia with hemiplegia from

which complete recovery took place. Associated with these were the features of Raynaud's disease. The patient died in a severe attack with pain in the right hand, gangrene to the elbow, and coma. Epilepsy has been reported in a great number of cases, and in one case ~~in my clinic,~~ reported by Thomas, the attacks only occurred in the winter when he had Raynaud's disease.

Albuminuria may occur during the attacks. Hæmoglobinuria has been present in a number of cases, and was well studied by the well-known surgeon, Druitt, in his own case. It is of the same nature as the paroxysmal hæmoglobinuria already described.

Scleroderma of the fingers may follow recurring attacks. Occasionally true generalized scleroderma begins with the features of Raynaud's disease. Arthritis has been present in certain cases.

Diagnosis.—There is rarely any difficulty in the diagnosis. One condition closely simulates it, namely, local gangrene of the toes associated with obliterative arteritis; but this occurs most frequently in older persons, in diabetic subjects, or in connection with well marked arterio-sclerosis. As a rule, the pulse in such cases is not to be felt in the dorsal artery. Allied to this form is a remarkable affection described by Brueger among the Russian Jews in New York—thrombotic phlebo-arteritis of the vessels of the leg with local gangrene. In the early stages the resemblance to Raynaud's disease is very close.

In the acute infections, particularly typhus fever, occasionally in epidemics of typhoid fever, and in malaria, areas of multiple gangrene occur. The distribution is usually different, and there is rarely any difficulty in distinguishing this form from Raynaud's disease.

Lastly, there are cases of multiple neurotic skin gangrene met with in hysterical and nervous patients, in the majority of which the lesions are self-inflicted. In military recruits local gangrene of the big toe has been caused by carbolic acid, and it seems probable that all of those so-called trophic and hysterical lesions are simulated.

Treatment.—In many cases the attacks recur for years uninfluenced by treatment. Mild attacks require no treatment. In the severer forms of local asphyxia, if in the feet, the patient should be kept in bed with the legs elevated. The toes should be wrapped in cotton wool. The pain is often very intense and may require morphia. Carefully applied, systematic massage of the extremities is sometimes of benefit. Galvanism may be tried. ~~Barlow advises immersing the affected limb in salt water and placing one electrode over the spine and the other in the water.~~ Nitroglycerin has been warmly recommended ~~by Cates~~. Calcium lactate in 15 grain (1 gm.) doses, three or four times a day, is sometimes very effectual. It often relieves chilblains. Cushing ~~has~~ introduced a plan of treatment with the tourniquet which has proved ~~very~~ successful in several cases ~~in my wards~~. The elastic bandage, or, better, ~~one of his~~ pneumatic tourniquets, is applied to an extremity tight enough to shut off the arterial circulation and left for some minutes. On releasing the constriction the member flushes brightly, owing to the vasomotor relaxation. The application in cases of severe spasm may have to be repeated at frequent intervals before the vascular constriction in the affected parts will be overcome, and the normal temperature and color return in them.

Buerger. Thrombo-angeitis obliterans.

-an acute inflammatory lesion of the
thrombosis.
probably due to infection

II. ERYTHROMELALGIA

(*Red Neuralgia*)

Definition.—"A chronic disease in which a part or parts—usually one or more extremities—suffer with pain, flushing, and local fever, made far worse if the parts hang down" (Weir Mitchell). The name signifies a painful, red extremity.

Symptoms.—In 1872 (Phila. Med. Times, November 23d), in a lecture on certain painful affections of the feet, Weir Mitchell described the case of a sailor, aged forty, who after an African fever began to have "dull, heavy pains, at first in the left and soon after in the right foot. There was no swelling at first. When at rest he was comfortable and the feet were not painful. After walking the feet were swollen. They scarcely pitted on pressure, but were purple with congestion; the veins were everywhere singularly enlarged, and the arteries were throbbing visibly. The whole foot was said to be aching and burning, but above the ankle there was neither swelling, pain, nor flushing." As the weather grew cool he got relief. Nothing seemed to benefit him. This brief summary of Mitchell's first case gives an accurate clinical picture of the disease. His second communication, On a Rare Vaso-Motor Neurosis of the Extremities, appeared in the Am. Jour. of the Medical Sciences for July, 1878, while in his Clinical Lessons on Nervous Diseases, 1897, will be found additional observations.

The disease is rare. The feet are much more often affected than the hands. The pain may be of the most atrocious character. It is usually, but not always, relieved by cool weather; in one of my cases the winter aggravates the trouble. In a few cases (Elsner, Dehio, Rolleston) the affection has been complicated with Raynaud's disease.

Mitchell speaks of it as a "painful nerve-end neuritis." Dehio suggests that there may be irritation in the cells of the ventral horns of the cord at certain levels. Excision of the nerves passing to the parts has been followed by relief. In one of Mitchell's cases gangrene of the foot followed excision of four inches of the musculo-cutaneous nerve and stretching of the posterior tibial. Sclerosis of the arteries was found. Of the 9 cases in which the local conditions were studied anatomically, the only constant change was a chronic endarteritis (Batty Shaw).

III. ANGIO-NEUROTIC ŒDEMA

(*Quincke's Disease*)

Definition.—An affection characterized by the occurrence of local œdematous swellings, more or less limited in extent, and of transient duration. Severe colic is sometimes associated with the outbreak. There is a marked hereditary disposition in the disease.

Symptoms.—The œdema appears suddenly and is usually circumscribed. It may appear in the face; the eyelid is a common situation; or it may involve the lips or cheek. The backs of the hands, the legs, or the throat may be attacked. Usually the condition is transient, associated perhaps with slight

gastro-intestinal distress, and the affection is of little moment. There may be a remarkable periodicity in the outbreak of the œdema. In Matas' case this periodicity was very striking; the attack came on every day at eleven or twelve o'clock. The disease may be hereditary through many generations. In the family whose history I reported five generations had been affected, including twenty two members. The swellings appear in various parts; only rarely are they constant in one locality. The hands, face, and genitalia are the parts most frequently affected. Itching, heat, redness, or in some instances urticaria, may precede the outbreak. Sudden œdema of the larynx may prove fatal. Two members of the family just referred to died of this complication. In one member of this family, whom I saw repeatedly in attacks, the swellings came on in different parts; for example, the under lip would be swollen to such a degree that the mouth could not be opened. The hands enlarge suddenly, so that the fingers can not be bent. The attacks recur every three or four weeks. Accompanying them are usually gastro-intestinal attacks, severe colic, pain, nausea, and sometimes vomiting. It is quite possible that some of the cases of Leyden's intermittent vomiting may belong to this group. The colic is of great intensity and usually requires morphia. Arthritis apparently does not occur. Periodic attacks of cardialgia have also been met with during the outbreak of the œdema. Hæmoglobinuria has occurred in several cases.

The disease has affinities with urticaria, the giant form of which is probably the same disease. There is a form of severe purpura, often with urticarial manifestations, which is also associated with marked gastro-intestinal crises, and it is interesting to note that Schlesinger has reported a case in which a combination of erythromelalgia, Raynaud's disease, and acute œdema occurred. Quincke regards the condition as a vaso-motor neurosis, under the influence of which the permeability of the vessels is suddenly increased.

The **treatment** is very unsatisfactory. In the cases associated with anæmia and general nervousness, tonics, particularly large doses of strychnia, do good. I have seen great improvement follow the prolonged use of nitroglycerin; and calcium lactate may be tried, in doses of 15 grains (1 gm.) thrice daily.

IV. PERSISTENT HEREDITARY ŒDEMA OF THE LEGS

(*Milroy's Disease*)

This remarkable condition, first described by Milroy of Omaha, is characterized by persistent œdema of the legs, without any traceable cause, or any constitutional features. It is a fairly common complaint, affecting males and females equally. As many as 22 persons in Milroy's series were affected among 97 in six generations; in Hope and French's series 13 to 42 persons in five generations. The œdema is strictly limited to the lower limbs and varies very slightly. In some instances there are remarkable acute attacks, with chill, fever, and increase of swelling. Except mechanically the condition does not seriously interfere with health.

Here may be mentioned a remarkable familial affection described by Edgeworth of Bristol (*Lancet*, July 22, 1911), of a general subcutaneous œdema. Of six infants born of healthy parents, all but one died within the first few months, with general œdema, following upon diarrhœa. The cases differ essentially from those of œdema neonatorum.

V. FACIAL HEMIATROPHY

A rare affection characterized by progressive wasting of the bones and soft tissues of one side of the face. The atrophy starts in childhood, but in a few cases has not come on until adult life. Perhaps after a trifling injury or disease the process begins, either diffusely or more commonly at one spot on the skin. It gradually spreads, involving the fat, then the bones, more particularly the upper jaw, and last and least the muscles. The wasting is sharply limited at the middle line, and the appearance of the patient is very remarkable, the face looking as if made up of two halves from different persons. There is usually change in the color of the skin and the hair falls. Owing to the wasting of the alveolar processes the teeth become loose and ultimately drop out. The eye on the affected side is sunken, owing to loss of orbital fat. There is usually hemiatrophy of the tongue on the same side. Disturbance of sensation and muscle twitching may precede or accompany the atrophy. In a majority of the cases the atrophy has been confined to one side of the face, but there are instances on record in which the disease was bilateral, and a few cases in which there were areas of atrophy on the back and on the arm of the same side.

Of the autopsies, Mendel's alone is satisfactory. There was the terminal stage of an interstitial neuritis in all the branches of the trigeminus, from its origin to the periphery, most marked in the superior maxillary branch.

The disease is recognized at a glance. The facial asymmetry associated with congenital wryneck must not be confounded with progressive facial hemiatrophy. Other conditions to be distinguished are: Facial atrophy in anterior polio-myelitis, and more rarely in the hemiplegia of infants and adults; the atrophy following nuclear lesions and sympathetic nerve paralysis; acquired facial hemihypertrophy, such as in the case recorded by D. W. Montgomery, which may by contrast give to the other side an atrophic appearance; and, lastly, scleroderma (a closely related affection), if confined to one side of the face. The precise nature of the disease is still doubtful, but it is a suggestive fact that in many of the cases the atrophy has followed the acute infections. It is incurable.

VI. SCLERODERMA

Definition.—A condition of localized or diffuse induration of the skin.

Varieties.—Two forms are recognized: the circumscribed, which corresponds to the keloid of Addison, and to morphœa; and the diffuse, in which large areas are involved.

The disease affects females more frequently than males. The cases occur most commonly at the middle period of life. The *sclerema neonatorum* is a different affection, not to be confounded with it. The disease is more common in the United States than statistics indicate. I saw 20 cases in sixteen years.

In the *circumscribed form* there are patches, ranging from a few centimetres in diameter to the size of the hand or larger, in which the skin has a waxy or dead-white appearance, and to the touch is brawny, hard, and inelastic. Sometimes there is a preliminary hyperæmia of the skin, and sub-

sequently there are changes in color, either areas of pigmentation or of complete atrophy of the pigment—leucoderma. The sensory changes are rarely marked. The secretion of sweat is diminished or entirely abolished. The disease is more common in women than in men, and is situated most frequently about the breasts and neck, sometimes in the course of the nerves. The patches may develop with great rapidity, and may persist for months or years; sometimes they disappear in a few weeks.

The *diffuse form,* though less common, is more serious. It develops first in the extremities or in the face, and the patient notices that the skin is unusually hard and firm, or that there is a sense of stiffness or tension in making accustomed movements. Gradually a diffuse, brawny induration develops and the skin becomes firm and hard, and so united to the subcutaneous tissues that it cannot be picked up or pinched. The skin may look natural, but more commonly is glossy, drier than normal, and unusually smooth. With reference to the localization, in Lewin and Heller's statistics in 66 observations the disease was universal; in 203, regions of the trunk were affected; in 193, parts of the head or face; in 287, portions of one or other of the upper extremities; and in 122, portions of the lower extremities. In 80 cases there were disturbances of sensation. The disease may gradually extend and involve the skin of an entire limb. When universal, the face is expressionless, the lips can not be moved, mastication is hindered, and it may become extremely difficult to feed the patient. The hands become fixed and the fingers immobile, on account of the extreme induration of the skin over the joints. Remarkable vaso-motor disturbances are common, as extreme cyanosis of the hands and legs. In one of my cases tachycardia was present. The disease is chronic, lasting for months or years. There are instances on record of its persistence for more than twenty years. Recovery may occur, or the disease may be arrested. One of my patients, with extensive involvement of the face, ears, and hands, improved very much. The patients are apt to succumb to pulmonary complaints or to nephritis. Arthritic troubles have been noticed in some instances; in others, endocarditis. Raynaud's disease may be associated with it. I have seen an instance of the diffuse form in which the primary symptoms were those of local asphyxia of the fingers, and in which, with extensive scleroderma of the arms and hands and face, there were cyanosis and swelling of the skin of the feet without any brawny induration. The pigmentation of the skin may be as deep as in Addison's disease, for which cases have been mistaken; scleroderma may occur as a complication of exophthalmic goitre.

The remarkable dystrophy known as *sclerodactylie* belongs to this disorder. There are symmetrical involvements of the fingers, which become deformed, shortened, and atrophied; the skin becomes thickened, of a waxy color, and is sometimes pigmented. Multiple calcareous nodules, not unlike tophi, but not uratic, may occur about the fingers. Bullæ and ulcerations have been met with in some instances, and a great deformity of the nails. The disease has usually followed exposure, and the patients are much worse during the winter, and are curiously sensitive to cold. There may be changes in the skin of the feet, but the deformity similar to that which occurs in the hand has not been noted. Some of the cases present in addition diffuse sclerodermatous changes of the skin of other parts. In Lewin and Heller's monograph

there are 35 cases of isolated sclerodactylism, and 106 cases in which it was combined with scleroderma.

The **pathology** ~~of the disease~~ is unknown. It is usually regarded as a tropho-neurosis, probably dependent upon changes in the arteries of the skin leading to connective tissue overgrowth. The thyroid has been found atrophied.

Treatment.—The patients require to be warmly clad and to be guarded against exposure, as they are particularly sensitive to changes in the weather. Warm baths followed by frictions with oil should be systematically used. ~~I~~ have tried ~~the~~ thyroid feeding thoroughly in the diffuse form. In one case the disease appears to be arrested; the patient ~~has taken~~ the extract for seven years. In a second case, after a year the face became softer, and there ~~has~~ ~~been~~ permanent improvement. In a case of quite extensive localized scleroderma the patches became softer and the pigmentation much less intense. Salol in 15 grain (1 gm.) doses three times a day is stated to have been successful in several cases.

VII. AINHUM

Here a brief reference may be made to the remarkable trophic lesion described by Da Silva Lima, which is met with in negroes in Brazil, Africa, India, and occasionally in the Southern States. It is confined to the toes, usually the little toe, and begins as a furrow on the line of the digito-plantar fold. This gradually deepens, the end of the toe enlarges, and, usually without inflammation or pain, the toe falls off. The process may last some years. Cases have been reported in America by Hornaday, Pittman, F. J. Shepherd, and Morrison.

SECTION XII

DISEASES OF THE LOCOMOTOR SYSTEM

A. DISEASES OF THE MUSCLES

I. MYOSITIS

Definition.—Inflammation of the voluntary muscles.

A primary myositis occurs as an acute, subacute, or chronic affection. It is seen in two chief forms—the suppurative and non-suppurative.

I. Suppurative myositis, known also as infectious myositis, is especially frequent in Japan, where, according to Miyake, some 250 cases have been reported; but he claims that some of these examples belong to other affections. Miyake personally saw 33 cases in Japan during twenty one months' practice, and took cultures from all but one of them. In 2 cases the results were negative, but in 27 a pure culture of the staphylococcus pyogenes aureus was obtained, while in another the streptococcus and in 2 more the albus with the aureus was grown. The malady may involve one or many muscles, and is usually sudden in its onset. There are also high fever and marked prostration. Subsequently abscesses occur in the indurated muscles, and pyæmia may ensue if the implicated muscles are not thoroughly evacuated.

II. Dermato-myositis.—An acute or subacute inflammation of the muscles of unknown origin associated with œdema and dermatitis. Steiner collected 28 cases from the literature and reported two cases from ~~my~~ clinic. The muscle inflammation is here multiple, and is associated with œdema and a dermatitis. The case of E. Wagner may be taken as a typical example. A tuberculous but well-built woman entered the hospital, complaining of stiffness in the shoulders and a slight œdema of the back of the hands and forearms. There was paræsthesia, the arms became swollen, the skin tense, and the muscles felt doughy. Gradually the thighs became affected. The disease lasted about three months. The post mortem showed slight pulmonary tuberculosis; all the muscles except the glutei, the calf, and abdominal muscles were stiff and firm, but fragile, and there were serious infiltration, great proliferation of the interstitial tissue, and fatty degeneration. In the case reported by Jacoby the muscles were firm, hard, and tender, and there was slight œdema of the skin. The cases usually last from one to three months, though there are instances in which it has been longer. The swelling and tenderness of the muscles, the œdema, and the pain naturally suggest trichinosis, and, indeed, Hepp speaks of it as a pseudo-trichinosis. The nature of the disease is unknown. Of the 28 cases collected by Steiner 17 died. The anatomical changes are those just mentioned as found in E. Wagner's cases. One of

[illegible]

Fibrosis. B.M.J. 1913. i. 343.
Llewellyn. Gout in etiology. 29% - pyuric in many.
pain. swelling. Local heat. hyperæmia = persistent
venous stagnation. aided infiltration (nodules) = congestion in dura tissue
Stockman. - patt. oedema, proliferation. fibroblasts to compact nodule.
periarteritis + endarteritis in b.v.s. of area
perhaps recessive. exudation of lymph & imperfect absorption.
Nathanson. heart-block found - maybe due to same process in
cardiac muscle

Senator's cases presented marked disorders of sensation and has been named neuro-myositis. Wagner suggests that some of these cases were examples of acute progressive muscular atrophy. The differentiation from trichinosis is possible only by removing a portion of the muscle. It has not yet been determined whether the eosinophilia described by Brown is peculiar to the trichinosis myositis.

III. Polymyositis Hæmorrhagica.—This form resembles the dermatomyositis in general features, but differs in the presence of hæmorrhages into and between the muscles. Of the ten cases analyzed by Thayer four recovered. Purpura and hæmorrhages from the mucous membranes may occur.

II. MYOSITIS OSSIFICANS PROGRESSIVA

This is a progressive inflammatory affection of the locomotor system of unknown origin, characterized by the gradual formation of bony masses in the fasciæ, muscles, aponeuroses, tendons, ligaments, and bones, with resulting ankylosis of most of the articulations (Steiner). About 100 cases have been reported. The process begins in the neck or back, usually with swelling of the affected muscles, redness of the skin, and slight fever, or with small nodules in the muscles which appear and disappear. After subsiding an induration remains, which becomes progressively harder as the transformation into bone takes place. The disease may ultimately involve a majority of the skeletal muscles. Nothing is known of the etiology. Malformation, microdactylism of the thumbs and big toes, is present in 75 per cent. of the cases.

III. MYALGIA

(*Fibrositis, Myositis*)

Definition.—A painful affection of the voluntary muscles and of the fasciæ and periosteum to which they are attached. It is probable that in many cases the fibrous tissue is especially affected—a fibrositis. It is by no means certain that the muscular tissues are the seat of the disease. Many writers claim that in some cases it is a neuralgia of the sensory nerves of the muscles. The affection has received various names, according to its seat, as torticollis, lumbago, pleurodynia, etc.

Etiology.—The attacks follow cold and exposure, and trauma is often a factor. It is most commonly met with in men, particularly those exposed to cold and whose occupations are laborious. It is apt to follow exposure to a draft of air, as from an open window in a railway carriage. A sudden chilling after heavy exertion may bring on an attack of lumbago. Persons of a gouty habit are certainly more prone to this affection, and one attack renders an individual more liable to another. It is usually acute, but may become subacute or even chronic, the last being more common in later life.

Pathology.—The changes are usually in the white fibrous tissue and are of an inflammatory nature. In acute cases there is a serous exudation in the affected parts and following this there may be proliferation of the fibrous

tissue. This may extend between the muscle fibres and cause stiffness and pain. Disability with muscular atrophy may result from this. Nodules sometimes form which may be painful.

Symptoms.—In the acute forms the affection is entirely local. The constitutional disturbance is slight, and, even in severe cases, there may be no fever. Pain is a prominent feature and may be constant or occur only when the muscles are drawn into certain positions. It may be a dull ache, like the pain of a bruise, or sharp, severe, and cramp-like. It is often sufficiently intense to cause the patient to cry out. Pressure on the affected part usually gives relief. As a rule, ~~myalgia~~ is a transient affection, lasting from a few hours to a few days, although occasionally it is prolonged for several weeks. It is very apt to recur.

Much attention has been given ~~of late~~ to a form of ~~myositis~~ occurring chiefly in the muscles of the head and neck, causing at first swelling and puffiness, later indurations. They are found particularly in the muscles at the back of the neck, but they are occasionally present in the muscles of the abdomen and limbs. They may appear quite suddenly, sometimes in muscles that are over-worked, but in other instances they seem to be ~~associated with~~ changes in the weather. The affection of the muscles of the head and neck may be associated with headache, the so-called indurative headache. Some of the cases have a picture very similar to migraine. In the abdominal muscles these limited swellings may cause pain and suggest appendicitis.

The following are the principal varieties ~~of myalgia~~:

(1) Lumbago, one of the most common and painful forms, affects the muscles of the loins and their tendinous attachments. Some patients are subject to attacks at short intervals. It occurs chiefly in workingmen and often after a strain in lifting. It comes on suddenly, and in very severe cases completely incapacitates the patient, who may be unable to turn in bed or to rise from the sitting posture. There may be difficulty in distinguishing this from arthritis of the spine or sacro-iliac joint disease. In these conditions the symptoms are more persistent. The restriction of motion in the spine is an aid in recognizing arthritis and the special features of sacro-iliac joint involvement should be looked for. The X-ray plates are a great aid.

(2) Stiff neck or torticollis affects the muscles of the antero-lateral or back region of the neck. It is very common, often unilateral, and occurs most frequently in the young. The patient holds the head in a peculiar manner turned to one side, and rotates the whole body in attempting to turn it.

(3) Pleurodynia involves the intercostal muscles on one side, and in some instances the pectorals and serratus magnus. This is, perhaps, the most painful form of the disease, as the chest can not be at rest. It is more common on the left than on the right side. A deep breath, or coughing, causes a very intense pain on pressure, sometimes over a very limited area. It may be difficult to distinguish from intercostal neuralgia, in which affection, however, the pain is usually more circumscribed and paroxysmal, and there are tender points along the course of the nerves. It is sometimes mistaken for pleurisy, but careful physical examination readily distinguishes between the two affections.

(4) Among other forms which may be mentioned are cephalodynia, affecting the muscles of the head; scapulodynia, omodynia, and dorsodynia, af-

Fibrositis in legs. tenderness spasm. increased by use = hammer toe
suggests. pain will float over. often a history of exposure to cold + damp.
Syphilis can be suspected elsewhere points. muddy clumsy flexor brain
digitorum or extensor tendons of great toe. Peri tendinitis is the rule
Fib. may extend to knee vessels and often varicose. Models may be put
in the sole.

Back. malignant disease or tuberculosis of bodies of lumbar vertebra = often suggested by occupation. Syphilis etc.

Ischaemic Lumbago. J.R. Hunt. J.A.M.A. 1914. 62, 611

lumbar type of rheumatoid arthritis. bicalcium uric acid.
clean white rest. warm cradle.
pain, cramp, but no nitrites rest of joints. pain radiates at times to
side or knee
are not tender, pain like without dependent on muscle exertion
bending moves acute pain.
Special nerves. are normal sphincters affected

Backache. Causes

1. Strain or Contr. of Ligaments — Strain in Lumbosacral region — occup. strains &c
bad posture — pelvic dis.

Backpain.

2. Inflammation — pneumo. — prost. neuro. vertb. — abscess in kid. Diabetes.

3. Infection — Vert. or intervert. joints — other arthritis — neuro. — tbc centres.

Causes — 1. Pelvic Disease —
2. Trauma Strain of muscl. joints
3. Arthritis
4. Static Lesion.
5. Sacro-iliac Strain
6. Sacro-iliac relaxation.

[illegible]

fecting the muscles about the shoulder and upper part of the back. Myalgia may also occur in the abdominal muscles and in the muscles of the extremities. The chronic forms are distinguished by soreness or pain associated with varying degrees of disability. There may be marked stiffness of the muscles, which are sometimes painful on pressure and may show definite tender areas of induration.

Treatment.—Rest of the affected muscles is of the first importance, and it is well to protect them from cold by a covering of flannel. Strapping of the side will sometimes completely relieve pleurodynia. No belief is more widespread among the public than in the efficacy of porous plasters for muscular pains of all sorts, particularly those about the trunk. If the pain is severe and agonizing, a hypodermic of morphia gives immediate relief. For lumbago acupuncture is, in acute cases, the most efficient treatment. Needles of from three to four inches in length (ordinary bonnet-needles, sterilized, will do) are thrust into the lumbar muscles at the seat of pain, and withdrawn after five or ten minutes. In many instances the relief is immediate, and I can corroborate fully the statements of Ringer, who taught me this practice, as to its extraordinary and prompt efficacy in many instances. The constant current is sometimes very beneficial. In many forms of myalgia the thermocautery gives great relief and in obstinate cases blisters may be tried. Hot fomentations are soothing, and at the outset a Turkish bath may cut short the attack. The bowels should be freely opened and large amounts of water taken. The salicylates are usually effectual; sodium salicylate (gr. x to xv, 0.6 to 1 gm.), aspirin (gr. x, 0.6 gm.), or salol (gr. v, 0.3 gm.) may be given. Some patients respond well to colchicum (♏ xv, 1 c.c. of the wine). In chronic cases potassium iodide may be used, and both guaiacum and sulphur have been strongly recommended. Persons subject to this affection should be warmly clothed, and avoid, if possible, exposure to cold and damp. In gouty persons the diet should be restricted and the alkaline mineral waters taken freely. Large doses of nux vomica are sometimes beneficial. Massage sometimes gives relief; it should be given gently at first and more vigorously later.

IV. MYOTONIA

(*Thomsen's Disease*)

Definition.—An affection characterized by tonic cramp of the muscles on attempting voluntary movements. The disease received its name from the physician who first described it, in whose family it existed for five generations.

While the disease is in a majority of cases hereditary, hence the name myotonia congenita, there are other forms of spasm very similar which may be acquired, and others still which are quite transitory.

Etiology.—All the typical cases have occurred in family groups; a few isolated instances have been described in which similar symptoms have been present. Males are much more frequently affected than females. In 102 recorded cases, 91 were males and only 11 females (Hans Koch). The disease is rare in America and in England; it seems more common in Germany and in Scandinavia.

Symptoms.—The disease comes on in childhood. It is noticed that on account of the stiffness the children are not able to take part in ordinary games. The peculiarity is noticed only during voluntary movements. The contraction which the patient wills is slowly accomplished; the relaxation which the patient wills is also slow. The contraction often persists for a little time after he has dropped an object which he has picked up. In walking, the start is difficult; one leg is put forward slowly, it halts from stiffness for a second or two, and then after a few steps the legs become limber and he walks without any difficulty. The muscles of the arms and legs are those usually implicated; rarely the facial, ocular, or laryngeal muscles. Emotion and cold aggravate the condition. In some instances there is mental weakness. The sensation and the reflexes are normal. G. M. Hammond has reported three remarkable cases in one family, in which the disease began at the eighth year and was confined entirely to the arms. It was accompanied with some slight mental feebleness. The condition of the muscles is interesting. The patients appear and are muscular, and there is sometimes a definite hypertrophy of the muscles. The force is scarcely proportionate to the size. Erb has described a characteristic reaction of the nerve and muscle to the electrical currents—the so-called myotonic reaction, the chief feature of which is that normally the contractions caused by either current attain their maximum slowly and relax slowly, and vermicular, wave-like contractions pass from the cathode to the anode.

The disease is incurable, but it may be arrested temporarily. The nature of the affection is unknown. Dejerine and Sottas have found hypertrophy of the primitive fibres with multiplication of the nuclei of all the muscles, including the diaphragm, but not the heart. The spinal cord and the nerves were intact. From Jacoby's latest studies it is doubtful whether these changes in the muscles are in any way characteristic or peculiar to the disease. J. Koch, however, has found, in addition to the muscle hypertrophy, degenerative and regenerative changes present, which he considers sufficient to account for the myotonic disorder. Karpinsky and von Bechterew, from careful urinary examinations, regard the affection as due to an auto-intoxication of the muscle tissue, caused by some faulty metabolism. No treatment for the condition is known.

V. PARAMYOCLONUS MULTIPLEX

(*Myoclonia*)

Definition.—An affection, described by Friedreich, characterized by clonic contractions, chiefly of the muscles of the extremities, occurring either constantly or in paroxysms.

The cases have been chiefly in males, and the disease has followed emotional disturbance, fright, or straining. The contractions are usually bilateral and may vary from fifty to one hundred and fifty in the minute. Occasionally tonic spasms occur. They are not accompanied by any sensory disturbances. In the intervals between the attacks there may be tremors of the muscles. In the severe spasms the movements may be very violent; the body is tossed about, and it is sometimes difficult to keep the patient in bed. Gucci has

56 autopsies since 1901 –
17 showed hyperplasia or persistence of thymus.
10 " thymic tumour – nearly all benign. (1 mal)

Sudden death may occur.

described a family in which the affection has occurred in three generations.

Weiss has also noted heredity in four generations. According to this author the essential symptoms are continuous or paroxysmal contractions, usually symmetrical and rhythmical, of muscles otherwise normal, which cease during sleep. There are neither psychical nor sensory disturbances. The condition is most common in young males, and is unaffected by treatment. Raymond groups this disease with fibrillary tremors, electric chorea (Dubini's disease), tic non douloureux of the face, and the convulsive tic, under the name of *myoclonies,* believing that it is only one link in a chain of pathological manifestations in the degenerate. Dana, in 1903, divided the myoclonias into five groups. In the first he placed paramyoclonus multiplex, and considered the names of four somewhat similar affections as synonyms of the same.

VI. MYASTHENIA GRAVIS

(*Asthenic Bulbar Paralysis; Erb-Goldflam's Symptom-Complex*)

Definition.—A disease with fatigue symptoms referable to the muscular system, due to failure of innervation without definite changes in muscles or nerves.

Of 180 cases collected by McCarthy, 83 were males and 96 females. The disease is most frequent in the third decade. The etiology is unknown.

The muscles innervated by the bulb are first affected—those of the eyes, the face, of mastication, and of the neck. After effort the muscles show fatigue, and if persisted in they fail to act and a condition of paresis or complete paralysis follows. All the voluntary muscles may become involved. After rest the power is recovered. In severe cases paralysis may persist. The myasthenic reaction of Jolly is the rapid exhaustion of the muscles, by faradism, not by galvanism. There are marked remissions and fluctuations in the severity of the symptoms.

Examination of the nervous system has revealed no abnormality. Weigert found a thymus tumor with metastatic growths in the muscles. Hun, Bloomer, and Streeter have described an infiltration of the muscles and of the thymus gland with lymphoid cells and a proliferation of the glandular elements of the thymus.

The diagnosis is easy—from the ptosis, the facial expression, the nasal speech, the rapid fatigue of the muscles, the myasthenic reaction, the absence of atrophy, tremors, etc., and the remarkable variations in the intensity of the symptoms. Of the 180 collected cases 72 proved fatal. The patient may live many years; recovery may take place. Rest, strychnia in full doses, massage, and alternate courses of iodide of potassium and mercury may be tried.

VII. AMYOTONIA CONGENITA

(*Oppenheim's Disease*)

A congenital affection characterized by general or local hypotonus of the voluntary muscles. Oppenheim called the disease *myatonia,* but this is pho-

netically so similar to *myotonia* (Thomsen's disease) that the name amyotonia of English writers is preferable.

Collier and Wilson, who have analyzed the recorded cases, give the following definition: "A condition of extreme flaccidity of the muscles, associated with an entire loss of the deep reflexes, most marked at the time of birth and always showing a tendency to slow and progressive amelioration. There is great weakness, but no absolute paralysis of any of the muscles. The limbs are most affected; the face is almost always exempt. The muscles are small and soft, but there is no local wasting. Contractures are prone to occur in the course of time. The faradic excitability in the muscles is lowered and strong faradic stimuli are borne without complaint. No other symptoms indicative of lesions of the nervous system occur."

In Spiller's case no lesions were found, but in an autopsy by Baudouin the cells of the anterior horns were found to be small, and there were extensive changes in the muscle fibres, similar to those in the myopathies.

B. DISEASES OF THE JOINTS

I. ARTHRITIS DEFORMANS

Definition.—A disease of the joints of doubtful etiology, but probably the result of infection, characterized by changes in the synovial membranes, cartilage, and peri-articular structures, and in some cases by atrophic and hypertrophic changes in the bones. A tendency to a chronic course is the rule.

Long believed to be intimately associated with gout and rheumatism (whence the names rheumatic gout and rheumatoid arthritis), this relationship seems disproved. By the studies of the Boston orthopedic surgeons (Bradford, Goldthwaite, and Lovett) and of Strangeways and his pupils at Cambridge (England) we are gradually getting a more accurate knowledge of the anatomical and clinical forms of this common disease. There is a difference of opinion as to whether there are two distinct diseases or varying forms of the same disease included under this heading. Those who hold the former view consider that in one disease the synovial membranes and the peri-articular tissues are particularly affected (rheumatoid arthritis) and in the other disease the cartilage and bone (osteo-arthritis). The disease occurs frequently and to it belong many of the cases termed "chronic rheumatism."

Etiology.—Age.—A majority of the cases are between the ages of twenty and fifty. In A. E. Garrod's analysis of 500 cases there were only 25 under twenty years of age. In 43 per cent. of the series of 350 cases studied by T. McCrae, the onset was before the age of thirty years. In the group with peri-articular changes predominating the age of onset is usually lower than in the group with special cartilaginous and bony changes.

Sex.—Among Garrod's cases there were 411 in women. More than half of the series of McCrae were males. The incidence as to sex is influenced by the inclusion of the cases of spondylitis, of which a large majority is in males. In women a close association with the menopause has been noted.

Predisposition.—In 216 in Garrod's series there was a family history of joint trouble; in McCrae's series only in 79 among 350. Two or three chil-

Kostrzewski, cinerary urn. Such suggest skin of deceased in many cases. perhaps used.

tuth absence

dren in a family may be affected. In America the incidence in the negro is relatively much less than in the white. Occupation and the station in life do not seem to have any special influence.

Exposure to cold, wet and damp, errors in diet, worry and care, and local injuries are all spoken of as possible exciting causes, but probably play but a small part.

As regards the views of the etiology of arthritis deformans, the one that it is of nervous origin is only of historical interest, and the modern opinion is that it is a result of some infectious process, certainly in the majority of cases.

Arthritis Deformans as a Chronic Infection.—In recent years this view has been gaining ground, although as yet positive bacteriological evidence is lacking. The infection may be with a specific organism or perhaps with various ones. Several workers have obtained organisms from the joints, and suggestive results have followed the injection of cultures in animals, but no constant association with any organism has been proved, and it seems probable that this form of arthritis may be secondary to infection with various organisms.

The acute onset, with fever in many cases, the polyarthritis, the presence of enlarged glands, the frequent enlargement of the spleen, the occurrence of pleurisy, endocarditis, and pericarditis in some cases are all suggestive of an infection. The likeness of the lesions to those due to arthritis from a specific cause, such as the gonococcus, is suggestive, and also the association of the arthritis with definite foci of infection in some cases. Lastly, a consideration of the form in children described by Still lends weight to this view, particularly in the widespread enlargement of the lymph glands and the swelling of the spleen. The sources of infection may be many: tonsillitis, suppuration in the nasal sinuses, chronic bronchitis, pyorrhœa alveolaris, pelvic disease in women and prostatic disease in men; the possibility of chronic infection from the intestinal tract must be considered. The infective theory of origin is gaining adherents, though it still lacks positive demonstration.

Metabolic.—While the nutrition suffers in many cases there does not seem any evidence to support the view that the disease is primarily due to disturbance of metabolism. Metabolic changes are probably secondary just as are the trophic changes.

Morbid Anatomy.—The usual descriptions are of the late stages of the disease when extensive damage has occurred, for there have been few opportunities to study the early changes, although more frequent operations have extended our knowledge of them and radiographs have aided much. There are three main forms of change: (1) Lesions principally in the synovial membranes and peri-articular tissues (the so-called rheumatoid arthritis), (2) with atrophic changes in the cartilage and bones predominating, and (3) with hypertrophy and overgrowth of bone (so-called osteo-arthritis). The first and second are seen most frequently in the joints of the extremities, the third in the spine. Whether these are distinct diseases or different manifestations of the same disease it is difficult to say. In many cases all forms of change are found, which speaks against the view that there are two distinct diseases. The changes in general are: (1) Effusion, which is not constant and shows no special features. (2) Changes in the synovial membrane. These are in-

flammatory and often hæmorrhagic at the onset. There may be marked thickening and proliferation of the synovial fringes with the formation of villi—villous arthritis. (3) The capsule and surrounding tissues may be infiltrated and much swollen. The peri-articular tissues show infiltration and swelling, and the enlargement of the joint is more often due to swelling about it than to bony changes. (4) Cartilage. This may show erosion, ulceration, atrophy, or proliferation. The cartilage may disappear entirely, but the changes are often very irregular and uneven and the cartilage may be replaced by fibrous tissue or by bone, the latter being most common at the edge of the cartilage. The cartilages may be soft and gradually absorbed or thinned (this often begins opposite the point of greatest involvement of the synovial membrane). (5) Bone. This may show atrophy of varying grade. If the cartilage is completely absorbed the surface of the bone often becomes hard and eburnated. In the form spoken of as *hypertrophic* there is new bone formation which is most common at the edge of the articular surfaces. In the hip joint this may form an irregular ring of bone about the joint cavity. The commonest example of overgrowth of bone is seen on the so-called "Heberden's nodes," which are bony outgrowths at the terminal interphalangeal joints. There may be deposit of new bone in the ligaments, particularly in the spine. Proliferation of bone usually occurs at the margins of the joints in the form of irregular nodules—the osteophytes—which may lock the joints. The formation of bone may also occur in ligaments, especially of the spine, which may be converted into a rigid bony column. Bony ankylosis rarely occurs in the peripheral joints, but is common in the spine.

There may be extensive secondary changes. Muscular atrophy is common and may appear with great rapidity. Subluxation may occur, especially in the knee and finger joints. The hands often show great deformity, particularly ulnar deflection. Contractures may follow and the joints become fixed in a flexed position. Neuritis and trophic disturbances may be associated; the neuritis is sometimes due to direct extension of the inflammatory process. Subcutaneous fibroid nodules occasionally occur.

The radiographs show the changes very well. Erosion of the cartilage is easily seen. In the type with predominant peri-articular changes the cartilage and bone often show little alteration. The occurrence of various changes in different joints or even in the same joint is common and bony change may occur with marked involvement of the peri-articular tissues.

Symptoms.—The onset may be acute or gradual. In the acute form a number of joints may be involved, there may be high fever and the whole condition be suggestive of rheumatic fever. In other cases the onset is acute in one joint and others are involved a few days later. With the gradual onset one joint is attacked and others follow. Some cases are between and may be termed subacute. In cases with an acute onset the attack may not persist very long; with the chronic onset the duration is usually prolonged. The acute onset occurs more frequently in the form in which changes in the soft parts predominate.

Arthritis.—In the acute form the joints are swollen, tender, and hot to the touch, but do not often show marked redness. There may be effusion in the larger joints. Pain is a marked feature and is increased by movement, the patient usually taking the position in which he has the greatest ease.

Osler - arthritis [illegible] - Llewellyn v Jones. 2 stages
1. villous hypertrophy for synovia. very [illegible]
2. true pathogenetic.

men - pseudoglisis magna very benign - bone not articulation

To diag. lesions plumbar spine from accessories.
patient supine - when knee flexed thighs on thorax side view
from table - (Sky)

When a joint is once attacked, the process does not subside quickly, and when the arthritis lessens some change remains in the joint which, however, may be very slight. The joints of the spine, especially in the cervical region, are often involved in the more acute forms, and in these there is rarely any permanent change. The temporo-maxillary joint is often involved, and arthritis here is always suggestive of this disease. The hands, when involved, show very characteristic changes. The knuckle joints are red, swollen, tender, and show limitation of motion. The fingers are often involved; swelling of the interphalangeal joints is common with a resulting thickening which gives a fusiform appearance to the finger. Partial dislocation, particularly at the terminal joint, is common. The knee joints are often involved with pain, effusion, limitation of motion, and later villous arthritis or subluxation. Thickening of the capsule usually occurs early.

In the hypertrophic (osteo-arthritis form) the process is rarely as acute as when the peri-articular parts are particularly involved (rheumatoid arthritis), but is usually polyarticular. The terminal finger joints, the hip joint, and the spine are especially affected. Pain is usually a marked feature, and the local features are not so marked. The process is more likely to be chronic.

Heberden's Nodes.—These are small bony outgrowths ("little hard knobs"—Heberden) at the terminal phalangeal joints, which develop gradually at the sides of the distal phalanges. They are much more common in women than in men. Heberden says "they have no connection with gout, being found in persons who have never had it," yet they are often regarded as indicating gout. In the early stage the joints may be swollen, tender, and slightly red, particularly when injured. The attacks of pain and swelling may come on in the joints at long intervals or follow injury. Sometimes they are the first manifestation of a general arthritis. Their distribution is not always regular and they are often largest on the fingers most used. They are often found in patients, the arthritis in whose other joints is of the other form. The condition is not curable; but there is this hopeful feature—the subjects whose arthritis begins in this way rarely have severe involvement of the larger joints. They have been regarded, too, as an indication of longevity.

The mon-articular form affects chiefly old persons, and is seen particularly in the hip and shoulder. It is identical with the general disease in its anatomical features. The muscles show wasting early and in the hip the condition ultimately becomes that described as *morbus coxæ senilis*. These cases seem not infrequently to follow an injury. They differ from the polyarticular form in occurring chiefly in men and at a later period of life.

The Vertebral Form (*Spondylitis*).—This may occur alone or with involvement of the peripheral joints. With the acute polyarthritis of the peripheral joints the spine may be involved, but there is usually no permanent change. With the hypertrophic form there is often bony proliferation and some spinal rigidity results which may involve the whole spine or only a part; in the latter case the lower dorsal and lumbar regions suffer most frequently. The condition may not involve more than a few vertebræ. The features are as variable as in the peripheral joints and there may be repeated acute attacks or a steady progressive process. In the general spine involvement the ribs may be fixed, the thorax immobile, and the breathing abdominal. There

are two varieties of the general involvement which are sometimes regarded as special diseases. In one (von Bechterew) the spine alone is involved, and there are pronounced nerve-root symptoms—pain, anæsthesia, atrophy of the muscles, and ascending degeneration of the cord. Von Bechterew thinks it begins as a meningitis, leads to compression of the nerve roots, loss of function of the spinal muscles, atrophy of the intervertebral disks, and gradually ankylosis of the spine. In the other—Strümpell-Marie type—the hip and shoulder joints may be involved (spondylose rhizomélique), and the nervous symptoms are less prominent. Both appear to be forms of arthritis deformans, and should neither be regarded nor described as separate diseases. Spondylitis deformans is more frequent in males than in females, and trauma probably plays an important part in its etiology. Local involvement is particularly common in the lumbar region and may cause sciatica and a great variety of referred pains. Pressure on the nerve-roots cause great pain, paræsthesia, and atrophy of the muscles. Movement of the spine is usually restricted.

Arthritis Deformans in Children.—Some cases resemble closely the disease in adults, in others there are very striking differences. A very interesting variety has been differentiated by Still, in which the general enlargement of the joints is associated with swelling of the lymph glands and of the spleen. The onset is almost always before the second dentition, and girls are more frequently affected than boys. The symptoms complained of are usually slight stiffness in one or two joints; gradually others become involved. The onset may be acute with fever or even with chills. The enlargement of the joints is due rather to a general thickening of the soft tissues than to a bony enlargement. The limitation of movement may be extreme, owing to the fixation of the joints, and there may be much muscular wasting. The enlargement of the lymph glands is most striking, increases with fever, and may be general; even the epitrochlear glands may be as large as hazelnuts. The edge of the spleen can usually be felt below the costal margin. Sweating is often profuse and there may be anæmia, but heart complications are rare. The children look puny and generally show arrest of development.

General Features.—*Temperature.*—In the acute attacks this may rise to 102° or 103° F., but is frequently lower and often persists for weeks with a maximum about 100° F. The *pulse* is usually rapid in proportion to the fever, the most frequent range being from 90 to 110. Cardiac changes are found in a small proportion of cases. *Glandular enlargement* is common and may be general or especially marked in the glands related to the affected joints. The *spleen* is enlarged in some cases, the frequency being greater in the younger patients. *Subcutaneous nodules* occur in a few cases and are sometimes tender. The *blood* often shows a slight anæmia, which is not as marked as might be expected from the appearance of the patients. There is rarely much increase in the leucocytes and the differential count shows no peculiarity. The *urine* does not show any change of moment. The *skin* sometimes shows irregular areas of yellow pigmentation, especially on the face and arms. It may have a glossy appearance over the affected joints. Profuse sweating of the hands and feet is common. The *reflexes* are usually increased in acute cases and a return to normal is of good significance. They are sometimes

Ausgabe an acuti streptococcus infection /

absent. Muscular *atrophy* is common and sometimes advances very rapidly. It is most marked in the hands. Twitching of the muscles is not uncommon.

In some patients the bony atrophy is very marked. This is most common in females. In these disorganization of the joints occurs and the cartilage rapidly disappears. These cases usually progress rapidly downward. This atrophy is to be distinguished from that due to disuse.

Course.—*General Progressive Form.*—This occurs in two varieties, acute and chronic. The *acute* form may resemble, at its outset, ordinary rheumatic fever. There is involvement of many joints; swelling, particularly of the synovial sheaths and bursæ, but not often redness; there is moderate fever which is often persistent and may be from 99° to 100° F. for weeks. The pulse rate is usually high in proportion to the fever. In this form there may be repeated acute attacks, perhaps at intervals of years, or there may be repeated attacks in various joints. These usually leave definite changes, which may be slight at first, but tend to increase with subsequent attacks. Acute cases may occur at the menopause. Some cases progress very rapidly; they lose weight and strength; atrophy and arthritic deformity are marked; and they suggest a progressive septic process without suppuration.

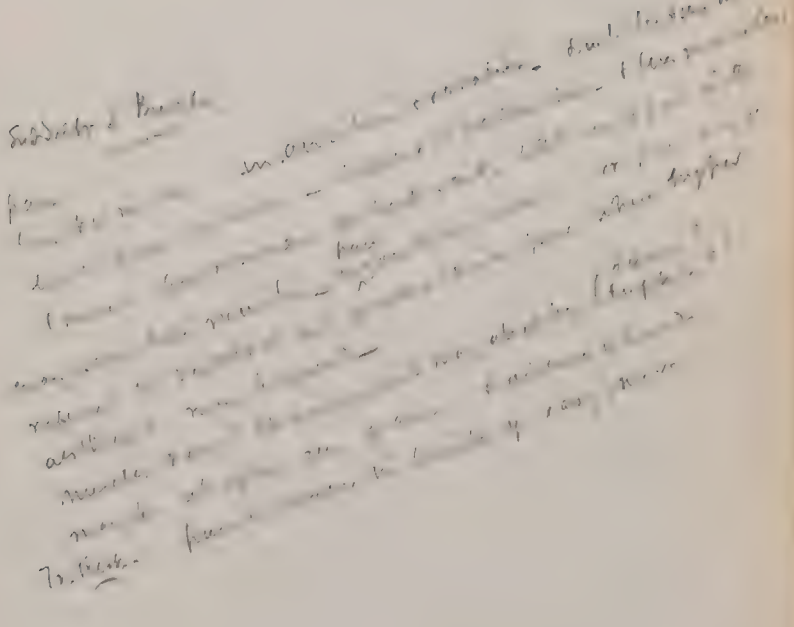
The *chronic* form is the most common, although most of these have had at some time an acute attack, especially at the onset. The first symptoms are pain on movement and slight swelling, which may be in the joint itself or in the peri-articular sheaths. In some cases the effusion is marked, in others slight. The local conditions vary greatly, and periods of improvement alternate with attacks of swelling, redness, and pain. At first only one or two joints are affected; gradually other articulations are involved, and in extreme cases every joint in the body is affected. Pain is an extremely variable symptom. Some cases proceed to the most extreme deformity without severe pain; in others the suffering is very great, particularly at night and during exacerbations of the disease. There are cases in which pain of an agonizing character is an almost constant symptom, quite apart from the occurrence of acute disturbances. Pain has an important influence in the production of deformity, as it hinders movement and the joints are kept in the position of greatest ease.

Gradually the shape of the joints is greatly altered, partly by the thickening of the capsule and surrounding tissues, perhaps by the pressure of osteophytes, and often by the muscular contraction. In moving the affected joint crepitus may be felt. Ultimately the joints may be completely immobile, not by a true bony anchylosis, although it may be by the osteophytes which form around the articular surfaces like ringbone in the horse, but more often from adhesions, and peri-articular thickening. There is often an acute atrophy of the muscles about the joints and atrophy from disuse usually supervenes, so that contractures tend to flex the thigh upon the abdomen and the leg upon the thigh. There are cases with rapid muscular wasting, symmetrical involvement of the joints, increased reflexes, and trophic changes, which strongly suggest a central origin. Numbness, tingling, pigmentation or glossiness of the skin, and onychia may be present. In extreme cases the patient is completely helpless, and lies with the legs drawn up, the arms fixed, and all the articulations of the extremities fixed. Fortunately, it often happens in these severe general cases that the joints of the hand are not so much affected, and the patient may be able to knit or write, though unable to walk or use the

arms. In many cases, after involving two or three joints, the disease becomes arrested, and no further development occurs. A majority of the patients finally reach a quiescent stage, in which they are free from pain and enjoy excellent health, suffering only from the inconvenience and crippling necessarily associated with the disease. Coincident affections are not uncommon. In the active stage the patients often suffer from dyspepsia, which may recur at intervals. A small percentage show cardiac lesions, and the pulse rate is usually higher than normal.

Diagnosis.—The cases with an acute onset may be difficult to distinguish from *rheumatic fever*. The affected joints are rarely as tender as in rheumatic fever, and the smaller joints are more often involved. The presence of thickening in a joint, rapid muscular atrophy, a relatively high pulse rate in relation to the fever (in the absence of endocarditis), and the absence of marked response to salicylate medication speak against rheumatic fever. The diagnosis from *gonorrhœal arthritis* may be difficult, but in this the small joints are usually not attacked so often, and after an onset with polyarthritis the majority of the affected joints usually clear, leaving one joint particularly involved. This rarely occurs in arthritis deformans. A careful search for gonococci is a great aid in diagnosis. In the chronic stage there may be considerable difficulty in distinguishing this disease from *gout*. This is particularly marked in either disease without marked joint changes. The study of the radiographs is particularly helpful and marked peri-articular changes speak for arthritis deformans. The finding of tophi or the estimation of the uric acid excretion may give the diagnosis of gout. It is important to distinguish *sub-deltoid bursitis* from the mon-articular form in the shoulder; the radiographs are a great aid. They are also important in the recognition of disease of the *sacro-iliac* joint and *tuberculosis* of the *hip-joint*. Special importance attaches to the diagnosis of the spinal forms. There is no difficulty in the case of general involvement, but with local changes in the lower spine it is not so easy. Pain on and restriction of movement are important; the patient is careful to limit any motion of the spine. Tuberculosis of the spine rarely offers any difficulty, especially with skiagrams.

Prognosis.—The age, general circumstances, character of the patient, the extent of damage to the joints, and the variety of change are all important. The outlook is not as dark as it is usually described. If the source of infection can be found and properly treated the prognosis is encouraging. In many patients the disease runs a certain course, and, if they can be brought through it with a minimum of damage, the ultimate outlook is good. In the form with peri-articular changes predominating, early diagnosis, treatment of the point of infection, the preservation of good nutrition, and a patient who is willing to fight are all encouraging factors. The outlook in the cases with the acute attacks is usually better than in those with a more chronic progressive course. Rapid muscular atrophy is of grave import. Cases in women beginning about the menopause should always have a grave prognosis. Rapid advancement in the joint changes is serious. In the form in children the outlook is not good, but some recover entirely. The group with marked hypertrophic changes (osteo-arthritis) usually do well. Heberden's nodes are permanent, but in the larger joints it is rare for the condition to advance to absolute crippling, although there may be considerable interference with function. Spondylitis



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rarely advances to complete immobility of the whole spine. The outlook is good in the local cases, but depends somewhat on the occupation and possibility of further trauma. The general condition of the patient is always of importance in estimating the outlook in any case. In those with marked nervous features the prognosis is not good.

Treatment.—Much depends on proper management and the pessimistic attitude is not justified. Certain things are important: early diagnosis so that treatment can be begun early, the avoidance of harmful measures, careful attention to the general condition, and every effort to limit the damage to the joints. Too much stress can not be placed on the need of early diagnosis; the disease is often regarded as "rheumatic" and the treatment directed to this (especially restriction of diet and the giving of salicylates for long periods) is usually harmful.

Source of Infection.—This should be carefully searched for and, if found, properly treated. If the tonsils are diseased they should be removed. Antistreptococcus serum has been used, and in some cases with benefit.

General Measures.—The patient should be kept out of doors as much as possible and every effort made to improve the general health. The *diet* should be the most nourishing possible. The mistake of cutting down the proteins is often made. Regard must be had to the digestion, and it is more often the carbohydrates which should be reduced. Water should be freely given, as elimination is important. The bowels should be kept open, and for this the salines are useful. It is important to see that the patients are warmly clad in cold weather and guarded against chilling. *Hydrotherapy* is useful locally in the form of compresses, but the hot bath treatment, so often given, more frequently does harm than good, particularly in acute cases. Baths, when taken, should be of very short duration. In more chronic cases bathing is sometimes of value. *Massage* is especially useful in the cases with synovial and peri-articular changes, and in them passive motion should be used early. *Climate* is of value in so far as patient is able to be out of doors and is saved from rapid changes of temperature.

Medicinal.—There is no drug which essentially influences the disease. The salicylates may be an aid in relieving pain, but should not be given for long periods. Iron, arsenic, and iodine are often useful. The iodide of iron in half drachm (2 c. c.) doses should be given persistently and alternated with Fowler's solution in five drop doses. Iodine may be given as the tincture in doses of two or three drops. Potassium iodide is sometimes of value when given for a long period. Thyroid and thymus gland extracts given persistently are sometimes beneficial. For the *pain* it is necessary to give drugs, although local measures should be used as much as possible. There are many which are available. Aspirin (gr. x, 0.6 gm.), guaiacol carbonate (gr. v, 0.3 gm.), antipyrin (gr. iii, 0.2 gm.), and sometimes codeia (gr. ½) are useful. Morphia should not be given on account of the danger of a habit.

Local.—(*a*) Use of the joints must be governed by the condition. When the cartilage and bones are not involved, passive motion and massage are useful, followed later by active motion. The patient should be taught simple exercises. When the cartilages and bones are involved, *rest* is usually advisable for a time. Every effort should be made to avoid contracture and displacement, and in this the use of splints during the night is often valuable.

Caution should be exercised in advising complete fixation. This is sometimes useful for short periods in the osteo-arthritic form, but may result in fixation in the other form and is usually not advisable for it. (*b*) *Counter-irritation.* This is usually an aid, and the Paquelin cautery, blisters, mustard, and iodine may be used. It is usually better to use light counter-irritation frequently than severe at longer intervals. (*c*) *Hyperæmia.* This may be active, and baking is a favorite method, but it should not be given for more than thirty minutes at a time. The temperature should be as high as the patient can stand. Passive hyperæmia may be used for a short period at first, and later for many hours at a time. (*d*) *Hydrotherapy.* The persistent use of compresses is often of value. They may be put on in the evening and left on all night.

Surgical Measures.—These are useful for the correction of deformities. In the case of villous arthritis operation is usually indicated. In the group with marked hypertrophy of bone removal of the outgrowths may be helpful.

Special Forms.—(*a*) *Heberden's nodes.* Avoidance of irritation and injury is important, and in the case of pain the use of compresses is helpful. (*b*) *Spondylitis.* During the acute stages rest is essential and should be secured by a plaster jacket or simple apparatus. In the milder forms firm strapping may give relief. Trauma should be especially avoided. (*c*) *Knee joint.* In many cases a simple elastic support is useful and may save the joint from injury.

Vaccines.—These have not proved of great value, but in a few cases the use of a polyvalent antistreptococcus serum has been of benefit.

Arthritis Secondary to Acute Infection.—While the majority of cases of arthritis are secondary to some form of infection, it is important to recognize that there are various forms. (1) Those with a definite bacterial cause, such as gonorrhœal or tuberculous arthritis. These usually have fairly well defined features. (2) Those secondary to infections of doubtful etiology, such as scarlet fever or measles. In some of these the arthritis is due to a secondary infection, but in others it appears to be due to the specific cause of the disease. (3) Arthritis secondary to definite infections in which there is no evidence of any organism in the joint. These are comparatively common and are difficult to designate. For example, arthritis, which may not be severe and subsides rapidly, occurs with an attack of tonsillitis or influenza. It has been suggested that these might be termed "toxic" or "toxæmic" arthritis. The term "infectious" arthritis, sometimes applied, is not a satisfactory one. The cases in this group usually clear without leaving permanent damage, but it is possible that if long continued they may result in the changes included under the heading of arthritis deformans.

"Chronic Rheumatism."—This term deserves mention because it is so commonly used, but it is a question whether its retention is justified. There is no uniformity in its usage and it is applied without discrimination to all kinds of arthritis and frequently to conditions which have nothing to do with the joints. Painful conditions of the joints, muscles, fasciæ, bones, and nerves are all termed "rheumatism." There is no disease entity to which the term can be applied, and it would be an advantage to give it up entirely.

II. INTERMITTENT HYDRARTHROSIS

The condition was described by Perrin in 1845, and there are about 70 cases on record (Garrod). The affection is characterized by a remarkable periodic swelling of one or several of the joints without fever. The swelling may take place with great rapidity, and there may even be a sensation of water rushing into the joint. There are usually pain and stiffness. The periods may be from ten to twelve days, or a month or even three months. Many of the cases have been in women and sometimes with marked hysterical symptoms. While some of the cases are secondary and only represent a phase in the evolution of various articular lesions, there appears to be a primary form characterized by a periodic swelling and nothing else. It is sometimes the joint equivalent to Quincke's œdema and may be associated with erythema, with angio-neurotic œdema, and in one of Garrod's patients there was at the same time circumscribed œdema of the lips and eyelids. A mother and daughter have been affected. The prognosis is not good; the attacks are apt to recur in spite of all forms of treatment.

C. DISEASES OF THE BONES

I. HYPERTROPHIC PULMONARY ARTHROPATHY

Definition.—A symmetrical enlargement of the bones of the hands and feet, and of the distal ends of the long bones, occurring in association with certain chronic diseases, particularly affections of the lungs.

Bamberger in 1889 reported a condition of abnormal thickening of the long bones in bronchiectasis, and the next year Marie described other cases and named the condition.

Etiology.—Clubbing of the fingers, or the Hippocratic fingers, represent a minor manifestation of this condition. Many varieties occur; indeed, there is a monograph with sketches of some thirty or forty forms. It is met with perhaps most constantly in congenital disease of the heart, in tuberculosis and in other affections of the lungs, particularly bronchiectasis, in congenital syphilis, in chronic jaundice, and in other chronic affections. In thoracic aneurism it may involve only the fingers of one hand. It usually comes on very slowly, but cases have been described of an acute appearance within a week or a fortnight. It may disappear. There is no bony alteration, but there is a fibrous thickening of the connective tissues with turgescence of the vessels. The condition is by no means easy to explain. The mechanical effect of congestion, the usual feature, explains the heart and lung cases, but not those of congenital syphilis and diseases of the liver, in which this is not present. Others have attributed it to a toxin.

Marie's syndrome is met with: (1) In diseases of the lungs and pleura. This was the case in 43 out of 55 cases collected by Thayer, and in 68 of Wynn's 100 cases. Bronchiectasis is the most common, then pulmonary tuberculosis and empyema.

(2) Other affections, such as chronic diarrhœa, chronic jaundice, nephritis, and congenital syphilis.

Marie regards the process as resulting from the absorption of toxins causing a periostitis; others have regarded it as a low form of tuberculous infection. The bones most frequently involved are the lower ends of the radius and ulna and the metacarpals, more rarely the lower end of the humerus, and the lower ends of the tibia and fibula.

Symptoms.—The affection comes on gradually, unnoticed by the patient. In other cases there is great sensitiveness of the ends of the long bones and of the fingers and toes. In one of ~~my~~ cases this was present in an extreme degree. The fully developed condition is easily recognized. The hands are large, the terminal phalanges swollen, the nails large and much curved. Similar changes occur in the toes, and the feet look large, especially the toes and the malleoli. The bones of the fore-arms are diffusely thickened, particularly near the wrist, and the tibiæ and fibulæ are greatly enlarged. Sometimes in advanced cases both ankles and knee-joints stand out prominently. The hypertrophy rarely affects the other long bones, though occasionally the extremities of the humerus and femur may be involved. The bones of the head are not attacked. Kyphosis may occur.

Diagnosis.—There is rarely any difficulty, as the picture presented by the hands and feet differs from that in acromegaly, and in practically all cases it is a secondary condition.

II. OSTEITIS DEFORMANS

(*Paget's Disease*)

Definition.—A chronic affection of the bones characterized by enlargement of the head, dorso-cervical kyphosis, enlargement of the clavicles, spreading of the base of the thorax and an outward and forward bowing of the legs.

The affection was described first by Sir James Paget, in 1877.

Etiology.—In the generalized form it is a rare disease, only two cases occurring among about 20,000 medical cases at the Johns Hopkins Hospital. ~~I have seen three or four instances in private practice.~~ The etiology ~~of the~~ disease is unknown. Mother and daughter have been affected. Some have regarded it as luetic, others as due to ~~the~~ arterio-sclerosis, which is a constant lesion. It may possibly be due to perversion of some internal secretion.

Pathology.—The skull, spine, and long bones are chiefly affected, those of the face, hands and feet are less involved. The skull may be as much as three quarters of an inch in thickness, and its circumference is increased. In one of Paget's cases it measured 71 cm. The shafts of the long bones are greatly thickened and they may weigh twice as much as a healthy bone of the same length. The femur is bent, the convexity forward; the tibiæ may be huge and very much bowed anteriorly. The bones of the upper extremities are less often involved, the spine shows a marked kyphosis, sometimes partial ankylosis; the pelvis is broadened.

The process is a rarefying osteitis which gradually involves the centre of the bones with the formation of Howship's lacunæ, Haversian spaces, and perforating canals. There is also new bone formation, both subperiosteal and myelogenous; the latter process gradually gains, and so the bones thicken.

Symptoms.—The disease begins, as a rule, in the sixth decade, sometimes

with rheumatoid pains, but more frequently the patient notices first that the head begins to enlarge, so that he has to buy a larger hat. Then his friends notice that he is growing shorter, and that the legs are getting more and more bowed.

There is a painful variety of the disease with great soreness of the arms and legs, which may be much worse at night. Headache, bronchitis, pigmentation of the skin, have been noted. The reduction in stature is very remarkable; one of my patients lost 13 inches in height.

Diagnosis.—The disease is readily recognized. The face differs from acromegaly, in which it is ovoid or egg-shaped with the large end down, while in Paget's disease the face is triangular with the base upward. In a few cases the disease may be limited to a few bones. There is a variety, of which I have seen three examples, involving the tibiæ and fibulæ alone, and in one to a slight extent the femurs. These bones gradually enlarge, are bowed anteriorly and laterally, so that the only obvious features are a reduction in height with bowing of the legs.

There is also a variety which is sometimes known as tumor-forming osteitis deformans, in which the bones are much deformed with multiple hyperostosis and new growths. The relation of this to Paget's disease is doubtful.

III. LEONTIASIS OSSEA

In a remarkable condition known as *leontiasis ossea* there is hyperostosis of the bones of the cranium, and sometimes those of the face. The description is largely based upon the skulls in museums, but Allen Starr has recently reported an instance in a woman, who presented a slowly progressing increase in the size of the head, face, and neck, the hard and soft tissues both being affected. He has applied to the condition the term *megalo-cephaly*. Putnam states that the disease begins in early life, often as a result of injury. There may be osteophytic growths from the outer or inner tables, which in the latter situation may give the symptoms of tumor.

IV. OSTEOGENESIS IMPERFECTA

(*Fragilitas Ossium*)

This is a systemic disease of the fetus in which the normal osseous development does not occur. At birth there is marked fragility of all the bones. There may have been intra-uterine fractures which have united and show large calluses. The extremities are often bent and deformed. The main features are defective development of the cranium and fragility of all the bones. It was thought that death always occurred, but Nathan has shown that some of the patients survive and that the bones become firmer as the child grows older. Treatment consists in using every protection against injury. Fractures usually unite readily.

V. OSTEOPSATHYROSIS

(*Fragilitas Ossium, Lobstein's Disease*)

Definition.—A rare affection characterized by abnormal brittleness of the bones.

In the aged the bones get thin and fragile, and the same happens in many chronic wasting diseases and in the insane. In osteopsathyrosis the general health of the patient is excellent and the fractures follow very trifling injuries, as a slight blow, or pull, turning over in bed, or in the simple act of chewing the patient may fracture the jaw. It is more common in the early period of life, and many of the cases in children represent the condition just spoken of—osteogenesis imperfecta—but there are cases in which it has continued until old age. The number of fractures which a patient may have may amount to one hundred or more. The fractures may be painless and heal readily; there are rarely any complications, and in a case reported by B. Sachs the condition was associated with general muscular atrophy and polyuria.

VI. ACHONDROPLASIA

(*Chondrodystrophia Fetalis*)

Definition.—A dystrophy of the epiphyseal cartilages due to connective tissue invasion from the periosteum, in consequence of which the epiphyses and diphyses are prematurely united and there is failure of the normal growth of the long bones. In consequence the subjects become dwarfs with normal heads and trunks, but short, stumpy extremities.

Description.—Achondroplasic dwarfs are easily recognized. They are well nourished and strong, and of average intelligence. Their height varies from 3 to 4 feet; the head and trunk are of about normal size, but the extremities are very short, the fingers, when the arms are at the sides, reaching little below the crest of the ilium. The important point in diagnosis is that in the shortness of the limbs it is the proximal segments which are specially involved, the humerus and femur being even shorter than the ulna and tibia (rhizomelia). The limbs are considerably bent, but this is more an exaggeration of normal curves and abnormalities in the joints than pathological curves as in rickets. The features of rickets are absent. The hand is short, and has a trident shape, since the fingers, which are of almost equal length, often diverge somewhat. The root of the nose is depressed, the back flat, and the lumbar lordosis abnormally deep, owing to a tilting forward of the sacrum. The scapulæ are short, the fibulæ longer than the tibiæ, and the pelvis is contracted; hence, the number of these cases reported by obstetricians. Heredity plays little part.

Pathology.—Anatomically it is a dystrophy of the epithelial cartilages, the cells of which are irregularly scattered, and the ground substances invaded by connective tissues from the periosteum, which sends in bands of

James A. Ballieu, Tyndall & Co. 1912. B.M.J. 1913. I. 305

Hereditary Deformity Chondrodysplasia. (Multiple Cartilaginous Exostoses)

see James Ehrenfried Feb 17, 17

" " " " 15, 15.

tissues across the end of the diaphysis. The development of the bones with a membranous matrix seems normal.

Virchow described the disease as fetal cretinism, others as fetal rickets. Of late naturally its origin has been associated with disturbance of the pituitary function, or of its hormonic relations. On the other hand, Murk Jansen of Leyden, in a recent monograph (1912), brings forward evidence to show that it results from a disturbance of the direct and indirect amniotic pressure, and he brings it into relationship with a number of other fetal malformations. He states, too, that the anatomical evidence is against changes in the sella turcica in the disease. But it is an argument in favor of some associated disturbance of the pituitary gland that achondroplasics often show precocious sexual development.

VII. OXYCEPHALY

Definition.—A cranial deformity associated with exophthalmos and impairment of vision.

Description.—The condition, known as *tower* or *steeplehead,* is characterized by great height of the forehead, sloping to a pointed vertex, with feebly marked supraorbital ridges, and the hairy scalp may be raised above the normal level, looking as if perched on the top of a comb. The intelligence is unimpaired. The condition is usually present at birth, though in some instances it develops from the second to the sixth year. As this curious growth of the head proceeds, headache may be present, exophthalmos develops, and the vision becomes impaired, due to progressive optic atrophy. Smell is often completely lost. The deformity appears to be due to premature synostosis of certain sutures, notably the sagittal and coronal. As a result of the premature union of these two sutures the growth of the vault of the skull is restricted in both its antero-posterior and tranverse diameters, and to accommodate the increasing bulk of the brain a compensatory increase in height takes place. Eventually the anterior fontanelle closes, but there is reason to think that this occurs at a later date than the normal, and its former site is marked by a slight protuberance with thinning of the bone. (Morley Fletcher, *Quarterly Jour. Med.,* IV, 1911.)

The optic neuritis and atrophy are the result of direct pressure exerted by the growing brain and may be compared to that of cerebral tumor. As yet we do not know the cause of this premature synostosis. The condition is one for which a decompression operation is indicated.

Lenticular Deg. hiccup.

with cirrhosis of liver.

Muscular weakness, dysphagia, nerve. atrophy. musc. twitchings

Cirrhosis - 4 cases. ascites 2. jaun. 1

Cirrhosis marked. f. [illegible] irregular

fracture at base of tube. Little portal cirrhosis

J. 4 cc. 8. Feb 17. 17

Cirrhosis - summary. 40 cases (Brissaud + Hallopeau) - 21 gen. hist

Skull fractures	1444	anaemia. 19.	Diabetes - 7
Cerebral hem.	95	cardiac. d. 14	tuberculosis 6
Meningitis.	26	delirium tr. 12	
pneumonia.	21	hepatic - 11	

Malig. affections - more common cases.

[illegible] 1175
1186

[illegible]

Dermographism = red line means irritability of vasodilator nerv.
White " " Spasm of vaso-constrictors "

Hyperaemia may come from (1) dilatation by irritable vaso-dil. "
(2) paralysis of vaso constrict. "
Skin reflex milestones which.

Mongolian Idiocy - syphilis suggested as cause -

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Vegetative nervous syst.

physiology. muscle & vessels of autonomic nervous system.

Sclera, iris & cil. sym. & vagus -

paralysis of bladder & colon with retention & helpd.

Symptoms & signs when bulbar segment of vegetative nervous system en-
tirely enlargements heart with retrosternal pain. asystole,
rapid pulse. dyspnoea. vomiting. & universal weakness. occ. cardiac
reg

mesencephalic segment - Drooping of lids. mydriasis &
pendulous pupil reaction.

Physiology tolerated by prob. & undetected [illegible]

Lehman - J.A.M.A. Feb. 27. 15. 782.

results not satisfactory. 95% of them except. to epinephrin use and
unexplained pilocarpine vice versa. In many who showed an
initial mydriasis no disturb. of veg. nerv. syst. manifest.
Others with vagus stigmata & reaction to pilocarpine had an
impulse from sympathetic - Suggests that whole veg. system & not vagus only.

Tests - to stimulate vagus - 0.01 gm. pilocarpine
" " sympathetic 0.001 or 0.00075 gm. [illegible]

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THE END

Pain produced by pressure is not accompanied by hyperesthesia of the
surrounding area (as rule)

Some exceptions — appendicitis
bursal pain with retroperitoneal trouble
Pelvic conditions
Sciatica & others.

Diagnostic.

Ask reflex — pat. on stem — legs in air — feel at right angles to leg.
Try to get Achilles reflex may fail or get bending of second
phalanx of big toe. Don't mistake if T. Ach. reflex is present.
occurs in injuries of cord, neurasthenia or sciatica.
Pat is complete section of sciatic — 2 abb. take good care.
Accompanied partially by Babinski process

4109234
v.2

not to circulate

MS.
Acc. no. 349
v. 2